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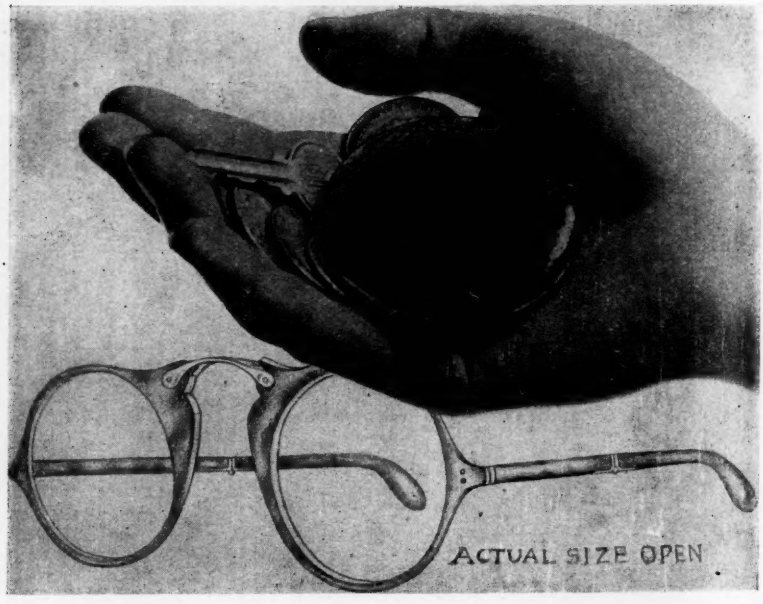

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THE BRITISH JOURNAL
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OPHTHALMOLOGY
JULY, 1930

COMMUNICATIONS

A RARE CONGENITAL ABNORMALITY OF THE EYE

BY
IDA MANN
LONDON

THE following case is reported, since, although it belongs to a group of congenital defects (fibro-lipoma, dermo-lipoma or dermoid of the cornea) which are relatively common and well known, it apparently furnishes an example of a very rare variety of the condition. As far as can be ascertained only three cases at all similar to the present have been described.

According to Stargardt[1] congenital abnormalities of the cornea of a fibro-lipomatous nature can be divided into four groups, which depend embryologically for their explanation on the period of development at which they arose. It appears from a survey of the literature that the occurrence of such anomalies is in inverse ratio to their earliness of origin. This is true of most congenital defects and is more or less self-evident, since the earlier in ontogeny that developmental aberration becomes evident the less good is the prospect of survival of the embryo.

All types of so-called congenital dermoid of the cornea can be referred to abnormal development of the mesoblast lying between the anterior rim of the optic cup and the surface ectoderm.

The first and rarest malformation can be referred to the period of growth immediately before the formation of the lens (in an embryo of about 4·5 mm.). Its definitive condition is characterised by the substitution for the cornea of a mass of fibrous tissue usually containing fat. The eye is always

microphthalmic and the membrane of Descemet, the anterior chamber, the iris and the lens are absent. As far as I am aware only three undoubted cases of this anomaly have been recorded. The present case affords a fourth example of this condition.

The cases in the second group arise during the process of formation of the lens, probably before its hyaline capsule is complete (*circa* 12 mm.). They show also the substitution of a mass of fibro-lipomatous tissue for the cornea, but this is associated with a poorly developed or even rudimentary lens. Traces of iris may be recognised in these cases.

In the third group the defect arises after the formation of the lens and anterior chamber and consists merely of the substitution of the abnormal mass for the whole or part of the cornea.

The fourth group contains the well-known limbal dermoids which are relatively common and arise as purely local anomalies of differentiation of a portion of the already forming cornea and sclera.

The present specimen will now be described and comparison made with the other recorded similar conditions.

I am indebted to Colonel J. N. Duggan of the Sir C. J. Ophthalmic Hospital, Bombay, for permission to publish the case. He has also kindly furnished me with the clinical history and the photographs of the child (Fig. 1.)

The child, a female Hindu of four months old, was brought to the hospital on January 25, 1927, on account of a congenital tumour of the left eyeball. The appearance of the child is shown in Fig. 1. In place of the left eyeball was a slightly movable pedunculated rounded swelling measuring about two inches in diameter. Posteriorly it was attached to a fibrous pedicle of about $\frac{3}{4}$ inch in diameter. The eyelids and eyelashes were normal. The former were found nearly encircling the stalk just behind the posterior surface of the tumour. The whole mass moved to some extent with the movements of the other eye, which was normal. On palpation the tumour felt hard. The surface covering resembled skin which passed imperceptibly into the conjunctiva covering the orbital cavity and the stalk. There was no sign or history of inflammation. The mass was removed under general anaesthesia. It could not be separated from the eyeball, of which it formed the major part. The stalk was ligated and cut. No abnormality in the orbital cavity was found. The child made an uninterrupted recovery.

The tumour was fixed in formol saline. It was divided in the sagittal plane. The pedicle, presumably the optic nerve, was attached to its posterior pole. The portion containing this was retained in the Pathology Department at Bombay. The other half

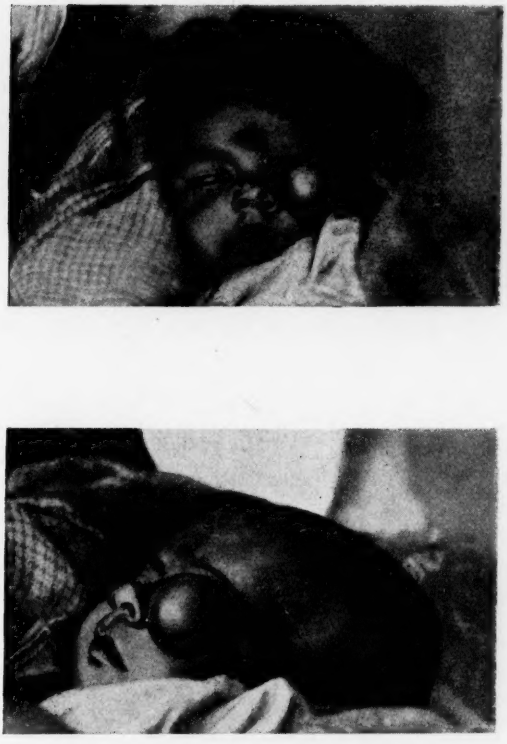

FIG. 1.

Appearance of the patient. (From a photograph by Colonel Duggan).

was brought to England by Colonel Duggan and sectioned in the laboratory at Moorfields.

The general topography of the specimen is shown in Fig. 2 which is three times the size after fixation. The actual specimen measured after fixation, 31·5 mm. × 25 mm. in its two greatest diameters.

The anterior portion of the mass (at A) is covered with skin. The mass itself is composed of fibrous tissue, containing areas of fat and in some situations showing myxomatous degeneration. This is most marked in the pale areas at M, which appear marked off by their less dense staining from the rest of the tumour. The posterior third of the tumour is occupied by a cavity, V, which represents the vitreous chamber. At the posterior pole, P, sclerotic

and choroid, normal in structure but rather thick, can be recognised. There is no trace of conjunctiva. The whole mass as far back as S is covered with pigmented skin. There is no trace of lens, anterior chamber, iris stroma or ciliary muscle. The neuro-epithelial elements derived from the primary optic vesicle are easily

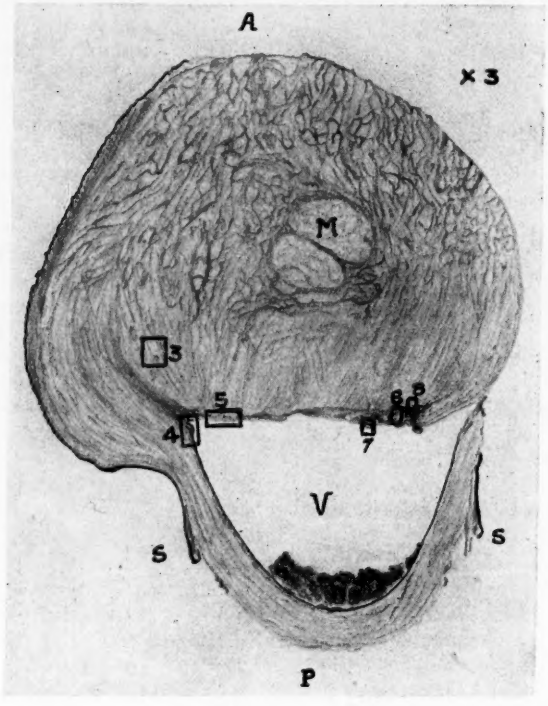

FIG. 2.

Gross morphology of the specimen. The rectangles 3, 4, 5, 6, 7, and 8 mark the situation of the sections shown in the subsequent figures.

recognisable, though they show aberrant development. It is much to be regretted that I was not able to obtain the pedicle for section. In the posterior part of the vitreous chamber there is a mass of blood clot, presumably operative and sub-retinal, and most of the retina has become detached and lost. Traces of it, showing an abnormally rich vascularisation, could be found in some of the sections.

The tumour is therefore obviously an aberrance of differentiation of the mesoblast lying between the optic cup and the surface ectoderm. It must have begun to arise very early, since it has inhibited the development of the lens entirely. The other accompanying abnormalities (skin instead of conjunctiva, faulty retinal differentiation and absence of iris) are secondary to this primary fault and can be explained as attempted adaptations to altered environment. Whether the mesoderm of the foetal intraocular vessels was also faulty cannot be determined from the portion of the specimen available, but it has been shown to be abnormally persistent in other cases of the same nature and it is possible that it was so in this case also, as there is evidence of excessive intraocular vascularity. No definite vitreous vessels were, however, recognised in either half.

Certain regions of the specimen are of special interest, some as showing the presence of abnormal tissues, others as illustrating altered growth in response to altered environment. (The small rectangles in Fig. 2 indicate the areas shown under higher magnification in subsequent figures.)

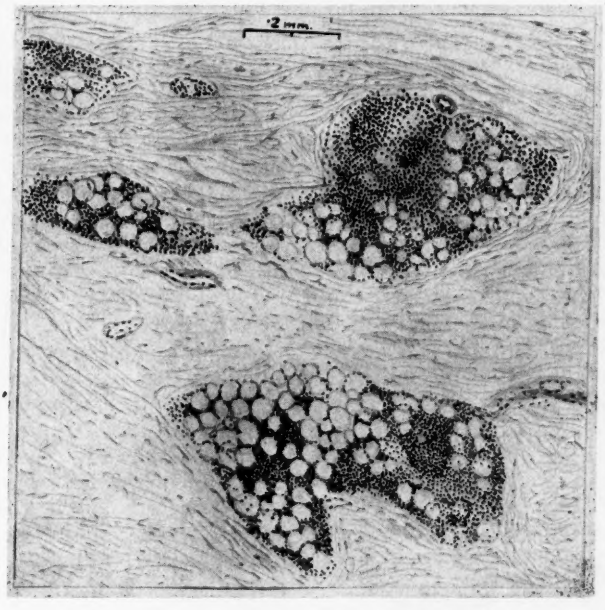

FIG. 3.

Fibro-lipomatous portion of tumour (at 3, in Fig. 2) showing cellular fat.

Immediately beneath the skin covering the surface of the tumour is a layer of fibrous tissue containing blood vessels and sweat glands. No hairs were seen. Deep to this again is a large area of fibro-fatty tissue similar to that found in any fibro-lipoma. At M the fibrous tissue predominates over the fat and shows myxomatous degeneration. Posterior to M there is still more fibrous tissue as compared with fat, and what fat there is is peculiarly cellular. Fig. 3 shows a portion of this cellular fat. It is lying in irregular spaces between bundles of rather primitive fibrous tissue containing blood vessels. The fatty tissue bears a very strong resemblance to bone marrow and when its cells are examined under a high power, a great diversity of form becomes apparent and it is possible to recognise with a fair amount of certainty the presence of myelocytes, megakaryocytes, basophile and eosinophile leucocytes, lymphocytes, normoblasts and free red cells. This myeloid reaction of the fat, though interesting, cannot be used, as might at first sight seem possible, to support any theory of teratomatous or blastomatous origin of the tumour, since it must be remembered that isolated islands of marrow-like tissue are of common occurrence in infants. They are found in the viscera, liver, spleen and kidney and in other situations in normal children and are merely an expression of the great haematopoietic capacity of young children. There is no *a priori* reason why they should not occur in a patch of atypically differentiated tissue as easily as in normal tissue.

The vagaries of the neural epiblast in relation to the posterior surface of the fibro-lipoma are worthy of study as they illustrate so well the pluripotentiality of the cells of the optic vesicle in early stages of development.

If the lining of the vitreous cavity be examined at 4 (in Fig. 2) it will be seen that in the situation where one might expect to find traces of ciliary processes there is instead a transition, in the outer layer of the optic cup, to a primitive type of retina. The fate of the inner layer cannot be traced, but the outer layer (*i.e.*, that which should have formed the anterior or outer layer of the epithelium) shows an interesting series of changes as one passes along the posterior surface of the tumour. Fig. 4 shows it forming a neuro-epithelium (like that found at the posterior pole in a 10 mm. human embryo) in which a marginal and a mantle layer can be recognised. 1 marks the nuclei of the mantle layer, 2 the marginal layer, 4 the point of transition from this to 5, the normal pigment epithelium. 3 is the fibrous tissue of the tumour. It is to be noted that, since the differentiation is taking place in the *outer* wall of the original optic cup the marginal layer is turned *away* from the vitreous cavity. As one proceeds along the posterior surface of the tumour one encounters a more advanced stage of differentiation. At 5 (Fig. 2) the tissue shows an inner and outer neuroblastic

layer such as is found normally at the posterior pole at 17-20 mm. Fig. 5 represents this. 1 is the " outer " neuroblastic layer, 2 the fibre layer of Chievitz, 3 the " inner " neuroblastic layer (in this abnormal situation " inner " and " outer " are of course reversed) and 4 an invasion by blood vessels from the fibrous mass. Between 4 and 6 (Fig. 2) there are large areas of very

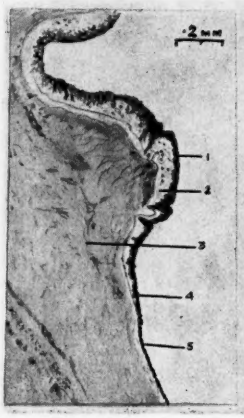

FIG. 4.

Abnormally differentiated retina. For reference numbers see text.

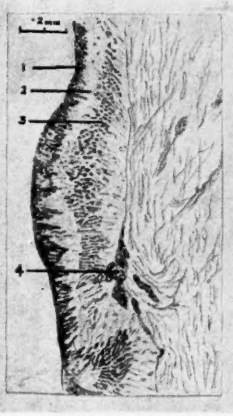

FIG. 5.

Abnormal retina showing inner and outer neuroblastic layers. For reference numbers see text.

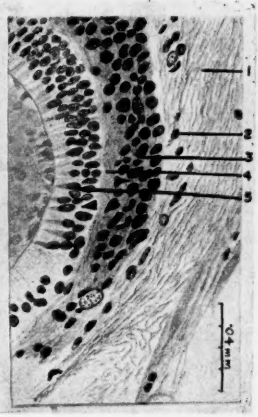

FIG. 6.

Abnormal retina showing three cell layers. See text.

atypical arrangement, but at 6 there is a small fold of tissue on which still further differentiation has occurred. Fig. 6 shows this. The outermost layer 1 is extremely fibrillar and represents the nerve fibre layer. 2 shows an irregular layer of isolated cells, some of which distinctly resemble ganglion cells. Deep to them at 3 is a layer of deeply staining nuclei (inner nuclear layer) separated by a narrow interval (4) from 5, a layer of large nuclei with open mesh and prominent centrosomes. These are oval, primitive in shape and are associated with curious large protoplasmic or cuticular protuberances towards and through a thin structureless membrane. The whole is reminiscent of the retina at 50-65 mm.; the rod and cone-like protuberances are larger than they would ever normally be, but their relation to the thin membrane (? external limiting membrane) is suggestive that they are morphologically comparable structures. They point, of course, towards the cavity of the primary optic vesicle.

Fig. 7 shows another atypical patch of tissue in which a somewhat primitive neuro-epithelium has undergone cystic degeneration. The patch of tissue is very reminiscent of the small retinal cysts often found at the ora serrata as a senile change in slightly degenerate eyes.

So far all the structures apparent in the neuro-epithelial tissue of the anterior part of the eye have been abnormal. It remains to

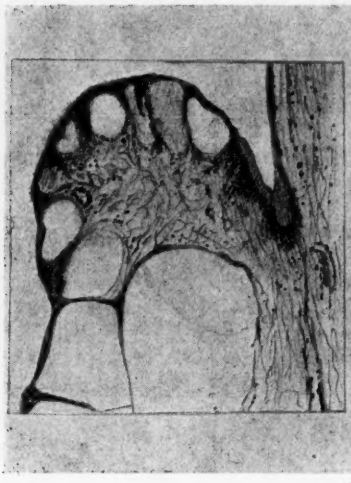

FIG. 7.

Cystic degeneration.

FIG. 8.

Neural tissue invading fibrous tissue. 1 points to neural epiblast, 2 to fibrous tissue.

ascertain whether any trace of the neural part of the iris, namely pigment epithelium or unstriped ectodermal muscle fibres can be discovered. Pigment is certainly present, but it occurs in isolated cells irregularly scattered among the abortive retinal elements and not anywhere as a definite layer. The question of unstriped muscle is more complicated. Sections stained with van Giesen's stain show (at 8 in Fig. 2 and for a short distance on either side) very clearly the distinction between the ectodermal and mesodermal elements in this region. The frankly retinal portion is easy to understand, but between it and the definite fibrous tissue is an irregular zone of yellow stained tissue markedly resembling unstriped muscle but directly continuous with the retinal portion. This tissue is invading the fibrous tissue and breaking it up into bundles. In places it appears syncytial, in others elongated cells with rod-shaped nuclei can be made out. It seems possible that

it represents an abortive attempt on the part of the neural ectoderm to form a sphincter. Fig. 8 shows it spreading into the fibrous tissue. 1 points to the neural tissue, 2 to the fibrous.

This case belongs to that group of congenital fibro-lipomata of the cornea which arise by an aberration of mesoderm at or about the fourth week of development. They are, according to Stargardt, true congenital anomalies (*Missbildung*) and should not properly be classed as new growths (*Geschwulst*). From a pathological point of view they belong to the group of monophyllic teratoblastomata as they arise primarily from the aberration of one germ layer only. The skin and abortive retina are merely changes in surrounding structures secondary to the primary anomaly. Such cases appear to be of extreme rarity. The three hitherto recorded which were undoubtedly of the same nature are those of Swanzy and Leber[2] (1871), Hanke[3] (1904), and Stargardt[1] (1917).

In Swanzy and Leber's case a large dermoid of the cornea was connected by a short thick pedicle with a further mass which projected outside the lids. At operation the eye was opened, with escape of vitreous. There was no trace of lens or anterior chamber. The abnormal fibrous tissue of the cornea blended with the iris. The presence of the constricted pedicle between the abnormal cornea and the pendulous extra-palpebral mass is explained easily as due to pressure on the base of the tumour by the growing lid folds which nip it. It affords a further piece of evidence of the very early date at which the anomaly arises, *i.e.*, before the lid folds are present (*circa* 18 mm.).

Hanke's case[3] is embryologically similar but clinically different as the mass was very small and did not project beyond the lids. The child was 14 years old. The mass of fibro-fatty tissue extended backwards in its upper part. The lens was absent. There were traces of iris especially below, where there was a small space possibly representing the anterior chamber.

Stargardt describes an extremely interesting case, clinically resembling Swanzy and Leber's[2] in that the tumour was in two parts united by a constricted pedicle. In this case two operations were performed, the tumour only being removed at the first, the rest of the eye subsequently. The iris, lens and anterior chamber were absent. There was no fat in the inside of the eye but the extra-palpebral portion of the tumour was a fibro-lipoma. One extremely interesting feature of the case was the fact that there was a persistent hyaloid running from the optic disc into the posterior surface of the corneal mass. A strand of vascular tissue containing nerve fibrils ran forwards from the anterior end of the hyaloid through both tumours and the intervening pedicle.

This connection of a persistent hyaloid with a fibro-lipoma of the cornea is also noted by Wagenmann[4] who described in 1889 a case

of fibro-lipoma of the cornea of the size of a hen's egg. At first sight this appears similar to the present case, but it shows the important morphological difference that rudiments of the lens were present, thus retarding the date of origin until after the 10mm. stage at least. Stargardt places this case in the first group but this is debatable in view of the presence of the lens.

The present case resembles that of Stargardt most closely though it is not possible on the material at hand to prove the persistence of the hyaloid artery. The tumour was probably not as large as Stargardt's and only shows a slight constriction round its base, which was embraced by the lids. The condition appears, however, to be of sufficient rarity to warrant its publication.

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THE SURGERY OF THE VITREOUS:

A preliminary paper dealing with an experimental Investigation into the Surgery of the Vitreous Body with special reference to its application in cases of Detached Retina, Primary Vitreous Disease, Haemorrhage or Traumatic loss of Vitreous.

BY

P. C. LIVINGSTON

SQUADRON LEADER, R.A.F.M.S.

History

The vitreous body, the very origin of which is yet open to speculation[1, 2], may in common with all living matter undergo changes both physiological and pathological that profoundly influence the vitality and function of its neighbouring structures. Between those delicate alterations of substance that produce the subjective symptoms associated with " muscae volitantes " on the one hand, to the sweeping havoc of a panophthalmitis on the other, lie conditions the ultimate termination of which are largely controlled by the state of the vitreous. A diseased vitreous body is a source of potential danger to sight both directly and indirectly, for in the first instance visual acuity must weaken when a hazy

medium has to be negotiated, while in the second place the disintegration of the supporting " gel " into a substance of little more than water makes the occurrence of such ophthalmic disasters as retinal detachment or haemorrhage mechanically more possible.

Regarding retinal detachment itself from a surgical standpoint no one operation at the present time can be said to hold out much help of permanent recovery although temporary benefit frequently results. The fact that so many surgical procedures are practised helps to strengthen the contention that none is reasonably successful.

De Schweinitz employs scleral puncture and salt injection. McMullen, repeated scleral puncture. Galezowski, a permanent drain with gold wire. Deutschmann performs a more drastic operation for diminishing the size of the eye globe itself; Müller excises some 20 mm. of sclera *via* Kronlein's approach, while Parker trephines and punctures and Meyer Weiner uses a horsehair drain. Nor is this list exhaustive, and each records successes: but no one form of technique stands out prominently as the operation of election unless perhaps it be cautery and puncture, or trephine and puncture. Regarding the use of cautery one writer[3], working experimentally, produces detachments by this means with a view to proving that the condition itself is the result of some change in the choroid whereby a post-retinal exudate is formed. On such grounds it would appear that the cautery as a means of cure is wrongly applied. Published results of the operative treatment of retinal detachment are confusing and variable. They range from something like 50 per cent. calculated successes, to all but complete failures and all in the hands of operators of repute. One authority, Vail (U.S.A.) in summarising the end results of a considerable number of cases studied by himself, comes to the unhappy conclusion that of these only some 20 may be maintained as representing permanent cures.

Object of this Paper

Approaching this discussion from another angle, we find studies of the vitreous body under normal and abnormal states dating so far back as 1741[4]; and coming nearer to the points at issue, researches concerning the effect on the eye of injections of foreign substances into the vitreous chamber[5]. The proposition at present under annunciation reveals an attempt to examine the subject of retinal detachment by dealing not only with the detachment as such, but also with the whole vitreous body should its state, from clinical observation, demand such procedure. The general principle of this vitreous surgery may prove of value also, where non-resolving haemorrhage, traumatic loss, or extensive primary disease makes such measures justifiable.

The Experiments

Seven dogs were made the subject of experiment in the investigation, which was designed to prove whether or not a tolerance existed, or could be without undue reaction established, between the vitreous of animals of the same species, and of animals of different species; and further, if such compatibility could be made out, whether vitreous retained in cold storage (for practical reasons) made any difference to the issue. The dogs were kept for varying periods after operation and the resulting series of ocular and constitutional changes are noted. At the conclusion of such time as was considered necessary for reactions to have reached their natural termination, the animals were destroyed. The eyes operated upon and in the case of the first four dogs, the unoperated eyes as well, were removed and prepared for section.

Technique

The technique employed was relatively simple and was not varied throughout the experiments. Vitreous was collected with adequate precaution against infection and injected into sterile test tubes which were immersed in a water bath at body heat. The period of times between obtaining the eyes (in the case of pigs and sheep) from the slaughter house and the extraction of the vitreous in the laboratory was on the average an hour—usually six eyes were used at a time so that sufficient vitreous might be to hand. Each eye in the case of pigs yielded 1·5 c.c. of vitreous without difficulty or complications such as the suction of lens or retinal substance into the syringe. The syringe was of the 5 c.c. " record " pattern, and had a needle of 2 mm. inside bore attached to it. A dog was anaesthetised (with chloroform and ether), the conjunctival sac washed out and a large speculum placed in position. Canthotomy was performed, and a long conjunctival incision was made parallel to the cornea and as near the equator of the globe as possible. A wide area of sclerotic was thus exposed and cleared.

Three fine silk sutures were taken and each in turn was made to enter and pass through the outer fibres of the sclerotic, twice, with a space of about 2 mm. intervening between the first and second insertion. This procedure gave a narrow strip of sclerotic controlled on either side by sutures. About 3 mm. separated one such suture from the next in vertical line. Owing to the extreme thinness of the dog's sclerotic (0·5 or 0·75 mm.), some difficulty was experienced in preventing complete perforation of the globe with the needles. Next, an incision was made down the line of guarded sclerotic. When the thickness of the sclerotic had been incised with a fine knife, a puncture opened choroid and retina and vitreous began to present. Next the needle with the syringe attached was

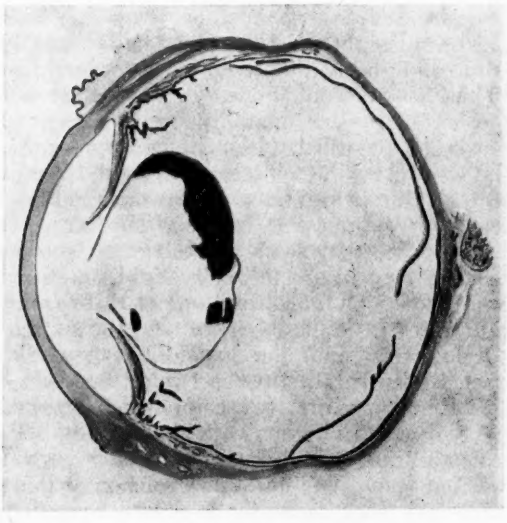
pushed well into the substance of the vitreous and 1 c.c. of it removed. After the sclerotic sutures had been knotted once and the edges of the wound drawn close about the needle, the syringe was now detached and another substituted which contained about 2 c.c. of foreign (*e.g.*, dog, pig, or sheep) vitreous, and this was then passed into the eye, digital pressure assuring the operator that the intra-ocular pressure did not rise too high.

It may be said here that sclerotic incision was found necessary, and no needle of any design could be made to perforate sclerotic tissue without undue pressure (7 and 9 oz.). When sufficient vitreous had been injected to leave the eye under normal conditions of pressure, the sutures were taken by an assistant and as the needle was withdrawn, tightened. By this means only a very slight vitreous loss was encountered, and the risk of losing an eye from " leakage " made exceedingly small. The wound of the conjunctiva was closed over in the ordinary manner.

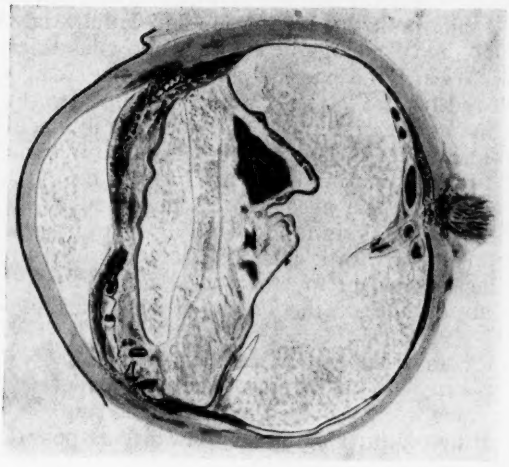
Clinical and Pathological Results

Of the seven dogs subjected to operation, three were treated with pigs' vitreous, one with sheeps' vitreous, one as a control with its own vitreous removed as described and replaced, and two with vitreous taken from other dogs. The attached table gives in brief the general reactions taking place from the time of the operation till the dogs were destroyed and the eyes removed. It will be observed that of the seven dogs operated upon, two eyes were lost, two retained useful sight, and three suffered visual loss (except for perception of light) without, however, experiencing marked reaction. These results would appear on the surface discouraging, and if representing the terminal product of experiment, should undoubtedly be so regarded. It must be appreciated, however, that these issues express in themselves merely the elementary stage in the process of this investigation; the work thus far being concerned solely with the question of *tissue tolerance*. Confined to such grounds we see that so far as tolerance is concerned, the several cases show up in a satisfactory light, in so much as two only of seven operations failed, and one of these from immediate haemorrhage which is beside the issue. Of the six remaining the only gross lesion appeared in the shape of one case of keratoconus, from protracted deep keratitis (Case 1), and one case of absolute glaucoma of secondary form (Case 5), which resulted from the intermingling of sheep vitreous with that of dog. Pig vitreous seemed reasonably inert while intermixing dog vitreous could be achieved without fear. The question of vision at this stage must be taken in a very open sense for it should be remembered that vitreous injected through a 2 mm. bore needle is broken by its passage into globules which reform imperfectly and will therefore

DOG 1. (Operated eye) 1 c.c. pigs' vitreous.



DOG. 3. (1 c.c. pigs' vitreous.) Vision satisfactory after twelve weeks.



DOG 5. Sheeps' vitreous 1 c.c. Destruction of eye from secondary absolute glaucoma.

cause very marked refractive changes. It is interesting in the circumstances to record two cases; in one of which vision was definitely good as tested by the ability of the animal to move easily among strange objects, and up a stairway, with the sound eye securely bandaged; while with the other dog the vision remained sufficiently good to permit these manoeuvres in a more restricted form. (Case 3 and Case 4.)

Pathologically the finding showed points of interest especially regarding the corneal changes. In no cases did the unoperated eye show alterations, either macro- or microscopical. The main features are summarised in the tables that have been drawn up and in the diagrams.

Field for future Investigation

If we can assume true compatibility between certain types of vitreous body, and the prospect of adequate vision under suitable operative technique, it remains then to perfect that technique by further experiment. Such a step demands the construction of a new type of syringe and needles of such bore that little damage may result to the vitreous substance during passage through them. Tests carried out on pigs' eyes have gone to show that the syringe should have a capacity of 10 c.c. and a minimum exit bore of 4 mm. The needle for the vitreous passage should have a 4 mm. internal bore and should be made oval in shape being thus 4mm. across its widest diameter, and 2·5 mm. across the smaller with fine

rubber tubing between it and the syringe. The operation suggested demands the simultaneous insertion and withdrawal of vitreous, the point of "*exit*" being the internal peri-equatorial area, and the point of "*inlet,*" the external peri-equatorial area. The needle to be employed for the "*exit*" would be bent at right angles so that it lies flat against the globe and does not hinder the internal ocular movements. Two fine eyelets welded to it would be available so that its position might be firmly fixed on the sclerotic by means of sutures. The needle to be employed at the "inlet" would have a similar design. Two syringes are necessary, braced in a common frame with a cross bar between them in order that they may be used either together or separately. With such an apparatus an outline of the operation suggested would be as follows:

(1) Attack first the detachment itself preferably by simple puncture or narrow bore trephine—and when the result is established (say three weeks) proceed with the next step.

(2) With the eye turned as far *out*wards as possible open the conjunctiva, retract it, and open the sclerotic between sutures (as described) adding two sutures for fixation through the eyelets in the needle. Pass and fasten the needle and attach rubber tubing.

(3) With the eye turned *in* as far as possible proceed as before, and fix the outer needle and tubing in place.

(4) Start by extracting vitreous up to 1 c.c. and then commence passing in the fresh vitreous from the outside syringe and continue until 5-6 c.c. have been inserted and removed.

(5) Close the wounds as described starting with the internal one.

Owing to other work the practical application of this technique has not been possible, but in view of the obvious stability found between certain types of vitreous it would seem a justifiable principle and one that might well be followed to its experimental conclusion.

I wish to thank Professor E. B. Verney, of University College, for his interest and ever ready help with this investigation; my colleague, Wing-Commander H. E. Whittingham, director of pathology R.A.F.M.S. for so kindly drawing the diagrams; and Messrs. Walls, of Chiswick, whose staff helped me considerably by providing at all times sufficient pigs' eyes to make the work smooth and easy.

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Pathological Findings

DOG 1. *Pigs'* vitreous 1 c.c. normal.

Cornea.—About double its thickness in parts. Epithelium intact. Substantia propria thickened and thrown into irregular waves chiefly the superficial layers directly behind Bowman's membrane. The fibres in this area are ill defined. Increase in number of corneal corpuscles. Increase of all elements immediately in front of Descemet's membrane. Endothelium in parts infiltrated by round and spindle-shaped cells.

Iris, lens, ligamentum pectinatum, clear.

Schlemm's Canal, clear.

Ciliary Body.—Some round-celled infiltration with disturbance of pigment cells. Migration of pigment cells.

Vitreous Body.—Areas of faintly staining material containing here and there scattered round cells. Migrated pigment cells. No leucocytes. No evidence (by oil immersion) of bacterial infection.

Optic Nerve and Retina.—Retina widely detached.. Appears normal until in region of nerve head. Here are again massed round cells and a band of tissue similar in appearance to the optic nerve projects well into the vitreous. It does not have the appearance of organised tissue. The unoperated eye shows no abnormal changes.

DOG 3. *Pig* 1 *c.c.*

Cornea.—One or two limited areas where the substantia propria is thrown into folds, and its continuity thereby broken. No definite cell invasion here and no thickening or alteration of the deeper layers. This condition can be seen to a very slight extent in sections of the normal eye and therefore may be explained as due in small part to the processes to which the eyes have been subjected in sectioning.

Iris, ciliary body, lens, vitreous and retina show no microscopical alterations.

The optic nerve is clear. (See diagram.)

DOG 4. *Dog into Dog.*

(Vitreous extracted and returned into same eye as control).

In the section of this eye and in those of the unoperated eye no abnormal changes can be noted.

DOG 5. *Sheep into Dog.*

Sections here show very profound reactions. Anterior chamber full of exudate and corneal distortion. Extensive iridic and ciliary body infiltration. Iris bombé. Destruction of lens. Marked vitreous reaction. Peculiar infiltration localised around nerve head, choroid and retina and lateral area. (See diagram.)

POST-OPERATIVE CLINICAL FINDINGS

Animal	Vitreous	Cornea	Lens	Vitreous	Retina and Disc	Vision	Duration of life after Experiment
(1) Dog	Pig 1 c c.	Early marked keratitis developing in 24 hours, reaching maximum point in 10 days and partially resolving in a month. Left some ectasia	Not seen	Not seen properly owing to keratitis	Condition unknown	Ability to detect light and darkness	Six weeks
(2) Dog	Pig 1 c.c.	Eye lost at operation from haemorrhage	—	—	—	—	—
(3) Dog	Pig 1 c.c.	Early slight keratitis. Clearing up on 3rd week and leaving a very faint haze in upper outer segment after 30 days	Nil abnormal	A few dark floating bodies of moderate size	A slight detachment at site of operation	Satisfactory. Could move around unknown objects without trouble (Normal eye bandaged)	Twelve weeks
(4) Dog	Dog (control) 1 c.c.	Nil abnormal	Nil abnormal	Nothing to note in this case	Did not appear affected	Satisfactory by the object avoiding tests	Eight weeks
(5) Dog	Sheep 1 c.c.	Haze from + tension after 7 days. No keratitis. Iris blurred	Not seen after 3 days	Not seen after 3 days	Did not appear affected	Blind	Ten days; destroyed owing to secondary absolute glaucoma
(6) Dog	Dog 1 c.c.	Nil abnormal	Normal	Some floating bodies. No other reaction noted	Some doubtful pallor of disc	Perception of light	Five weeks
(7) Dog	Dog 1 c.c. Cold storage 24 hours	Some transient haze lasting 4 days with change in iris pattern. Small hyphaema after 12 days, resolving 18 days	Normal	Some floating bodies	Nil abnormal	Perception of ligh	Five weeks

ON THE NATURE OF MILTON'S BLINDNESS

BY

ARNOLD SORSBY

LONDON

" . . . Thus with the year
Seasons return, but not to me returns
Day, or the sweet approach of even or morn,
Or sight of vernal bloom, or summer's rose,
Or flocks, or herds, or human face divine;
But cloud instead, and ever-during dark
Surrounds me, from the cheerful way of men
Cut off, and for the book of knowledge fair
Presented with a universal blank
Of Nature's works to me expunged and rased,
And wisdom at one entrance quite shut out."

PARADISE LOST. III. 40-50.

THE biographical and medical interest in the cause of Milton's blindness is evident; but it is not only the biographers and oculists who have been involved in this problem. Literary critics have struggled with this question and though there has been much speculation as to the effect of Milton's blindness on his later work, much ingenuity has also been expended in tracing the influence of his eyesight on his earlier writings.

I. The Evidence

The evidence available as to the nature of his blindness comes mainly from scraps of autobiographical detail left by Milton; these are supported and extended by the early biographies extant. That his blindness came on rather early in life is well known from the opening lines of the sonnet on his blindness:[1]

" When I consider how my light is spent
Ere half my days "

Some clue as to its nature is found in the other sonnet dealing with his blindness, the one addressed to his friend Cyriack Skinner.[2]

" Cyriack, this three years' day these eyes, though clear,
To outward view, of blemish or of spot,
Bereft of light, their seeing have forgot;"

That his eyes though blind were " clear to outward view, of blemish or of spot," is again asserted by Milton in his prose writings. Answering a personal attack he says[4] that but for his eyes he has not changed in any respect and as for these they " are externally uninjured. They shine with an unclouded light, just like the eyes of one whose vision is perfect. This is the only point in which I am against my will, a hypocrite."

A further reference to his eyes is found in " Paradise Lost." In a glorious paean on light[3] he says :

" Thee I revisit safe,
And feel thy sovran vital lamp ; but thou
Revisit'st not these eyes, that roll in vain
To find thy piercing ray, and find no dawn ;
So thick a drop serene hath quenched their orbs,
Or dim suffusion veiled."

Drop serene and dim suffusion are not so much alternate diagnoses as different names for very much the same thing, for *gutta serena* was the name for blindness with a transparent (*i.e.*, non-obscured) pupil* and *suffusio nigra* stood for blindness in which the pupil was black (and not grey). Both these names, therefore, agree with the statements made by Milton elsewhere to the effect that in his blindness his eyes were normal in appearance. Milton has also left us information on the state of his eyes during his childhood, and expressed the opinion that his early studies had harmed his sight. On this subject he says :[4]

" From the twelfth year of my age I scarcely ever went from my lessons to bed before midnight; which indeed was the first cause of injury to my eyes, to whose natural weakness there were also added frequent headaches." That excessive work led to his blindness was the belief of himself and his medical advisers. He records the circumstances under which he undertook to write a defence of of the republican ideal :[4]

" . . . . When I was publicly solicited to write a reply to the defence of the royal cause ; when I had to contend with the pressure of sickness and with the apprehension of soon losing the sight of my remaining eye; and when my medical attendants clearly announced that if I did engage in the work, it would be irreparably lost—their premonitions caused no hesitation and inspired no dismay." Indeed Milton found the yoke of blindness tolerable through his belief that he sacrificed his eyes in the service of his ideal :[2]

" Yet I argue not
Against Heav'n's hand or will, nor bate a jot
Of heart or hope, but still bear up and steer
Right onward. What supports me, dost thou ask ?
The conscience, friend, to have lost them overplied
In Liberty's defence."

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* " Yet the more common name (for cataract) among the Arabians was gutta obscura, as that for amaurosis was gutta serena ; the pupil in the last species being serene or transparent.

The Arabians, who had adopted generally the humoral pathology of Galen, conceived both these diseases to be the result of a morbid rheum or defluxion falling on a particular part of the visual orb, in one case producing blindness with obscurity, whence the name of an obscure rheum or gutta, and in the other without obscurity, whence the contrary name of a transparent or serene rheum or gutta.

But as various other diseases and particularly of the joints were also supposed to flow from a like cause, and were far more common, the term gutta and rheuma were afterwards emphatically applied and at length altogether limited to these last complaints: whence the terms gout and rheumatism." " The Study of Medicine," by J. M. Good, Vol. III, p. 219, London, 1822.

Most important information of the nature of his blindness is preserved for us in a letter written by Milton[5] to his friend Philaras, a Greek diplomat who had asked Milton to give him an account of his blindness, so that he might consult Dr. Thevenot, a Parisian oculist of great repute. What Thevenot did, or indeed whether the letter reached him is not known. In this letter Milton gives an account of the subjective symptoms he experienced during the development of his blindness. The letter is dated September 28, 1654, and by this time Milton had been blind for about three years.

" It is ten years, I think, more or less since I felt my sight getting weak and dull, and at the same time my viscera generally out of sorts. In the morning if I began as usual, to read anything, I felt my eyes at once thoroughly pained, and shrinking from the act of reading, but refreshed after moderate bodily exercise. If I looked at a lit candle, a kind of iris seemed to snatch it from me. Not very long after, a darkness coming over the left part of my left eye (for that eye became clouded some years before the other) removed from my vision all objects situated on that side. Objects in front also, if I chanced to close the right eye, seemed smaller. The other eye also failing perceptibly and gradually through a period of three years, I observed some months before my sight was wholly gone, that objects I looked at without myself moving, seemed all to swim, now to the right now to the left. Inverterate mists now seem to have settled in my forehead and temples, which weigh me down and depress me with a kind of sleepy heaviness, especially from meal-time to evening. [Here follows an allusion to classical literature]. But I should not forget to mention that, while yet a little sight remained when first I lay down in bed, and turned myself to either side, there used to shine out a copious glittering light from my shut eyes; then that as my sight grew less from day to day, colours proportionately duller would burst from them, as with a kind of force and audible shot from within; but that now, as if the sense of lucency were extinct, it is a mere blackness or a blackness dashed, and as it were interwoven with an ashy grey, that is wont to pour itself forth. Yet the darkness which is perpetually before me, by night as well as by day, seems always nearer to a whitish than to a blackish, and such that, when the eye rolls itself, there is admitted, as through a small chink a certain little trifle of light."

It is worth noting that this document so careful in its details is confined entirely to subjective symptoms: the physical appearance of his eyes is not touched upon. In view of the fact that this letter is a personal statement intended for an oculist it is hardly likely that Milton would have suppressed any obvious external change in the appearance of his eyes, and this letter must therefore be taken as lending further support to his statements elsewhere that his eyes were free from blemish or spot.

There is therefore no reason to doubt the diagnosis of *gutta serena* which Milton gives of his case in " Paradise Lost."

Apart from these autobiographical details there is some further evidence bearing on the subject in the contemporary portraits and sketches of Milton and in the lives of him by his contemporaries.

As for the portraits and sketches, there is unfortunately little of any importance to be obtained in this direction. Though very many etchings are available which are claimed to represent Milton at some time or other, it would appear that there are only two undoubted contemporary portraits.[10] Obviously but little significance can be attached to copies of later dates, and in so far as the present writer has seen them they are entirely negative for the appearances of the eyes in all of them are normal. The two undoubted portraits of Milton have been inaccessible to the writer but good photographs of them show nothing abnormal about the eyes. The subject of Milton's portraits is a hightly complicated one and open to considerable dispute[9]—and to draw positive conclusions from any of them would be a procedure of doubtful wisdom. All that can be said is that there is nothing in the available representations of Milton to disprove his statement as to his eyes during his blindness, and presumably earlier on, being of normal appearance.

The evidence as to Milton's appearance and his mode of life as retailed by biographers who were his contemporaries are in the main but confirmations of his own autobiographical notes dispersed through his writings. Thus the earliest " Life of Milton "[6] extant, generally ascribed to his friend and physician Nathan Paget, tells of his " sitting up constantly at his study till midnight " in his early years. The onset of his blindness is thus related:

" Whilst he was thus employed* his eyesight totally failed . . . from a weakness which his hard nightly study in his youth had first occasioned and which by degrees had for some time before deprived him of the use of one ey: And the Issues and the Seatons made use of to save or retrieve that, were thought by drawing away the Spirits, which should have supply'd the optic vessells, to have hastened the loss of the other. Hee was inded advised by his Physitians of the danger in his condition, attending so great intentness as that work required."

The following extract is of interest: " He was of a moderate stature, and well proportioned, of a ruddy Complexion, light brown Hair and handsome Features; save that his Eyes were none of the quickest. But his blindness, which proceeded from a Gutta

* In writing the Second Defence.

Serena, added no further blemish to them.* His deportment was sweet and affable, and in Gate erect and Manly . . . on which account hee wore a Sword while hee had his Sight, and was skilled in using it."

Milton's later biographers add but little to these pen-pictures. In fact the later Lives appear to be based on this first Life and the details supplied by Milton. One noteworthy remark appears in the Life by Aubrey,[8] who knew Milton personally. Says Aubrey:

" His father read without spectacles at 84, his mother had very weak eies and used spectacles presently after she was thirty years old."

Of the other contemporaries who have written about Milton, none throws any further light on the subject; but the following remark taken from the Life by his nephew Edward Philips[7] shows that the resignation with which Milton took his blindness, when at length it fell on him, was not the spirit in which he took the catastrophe during the long years it was preparing.

" . . . . His sight, what with his continual study, his being subject to head-ake, and his perpetual tampering with physick to preserve it, had been decaying for about a dozen years . . . the sight of one for a long time clearly lost."

The information obtained from the original sources as to Milton's eyesight and the possible causes of his blindness may therefore be summarised as follows:

(1) Milton's eyes had a " natural weakness." " His eyes were none of the quickest."

(2) His vision in his early days could not have been defective to a very high degree for " Hee wore a Sword while he had his Sight, and was skilled in using it." " I was won't constantly to exercise myself in the use of the broad sword, as long as it comported with my habits and my years."[4]

(3) The exact date of his blindness is not known with certainty. By 1654, the year that Milton wrote to Philaras the letter giving details of his case, he speaks of his blindness as though it were not a recent calamity. In a state paper, a letter from the Hague dated June 20, 1653, Milton is spoken of as being blind. The writer of " Regii Sanguinis Clamor " which was published in 1652 makes a personal attack on Milton, upbraiding him with his blindness selecting for the motto of his work Virgil's description of the eyeless cyclops: *Monstrum horrendum, informe, ingens, cui lumen ademptum.*—A monster horrid, hideous, huge and blind. Masson[11] holds that Milton's blindness was complete by March or April, 1652—and this date is generally accepted.

* Taken in its context the reference to Milton's eyes being none of the quickest seems to imply an anatomical rather than a physiological defect. But all the evidence is against such a reading.

(4) Milton was therefore only 43 at the time of his blindness, and from his letter we know that his sight had been " getting weak and dull " for about ten years before 1654, that is from his 35th year.* " Not very long after a darkness coming over the left part of my left eye (for that eye became clouded some years before the other) removed from my vision all objects on that side." It would therefore appear that the left eye had failed when Milton was not very much more than 35. The right eye does not seem to have failed so suddenly. " The other eye failing perceptibly and gradually through a period of three years. I observed some months before my sight was wholly gone, that objects I looked at without myself moving, seemed all to swim."

(5) Milton's blindness seems not to have been associated with any external signs; the eyes " are externally uninjured. They shine with an unclouded light, just like the eyes of one whose vision is perfect."

(6) Milton's " father read without spectacles at 84, his mother had very weak eies and used spectacles presently after she was thirty years old." In addition there is also the account of the subjective sensations felt by Milton in the terminal stages of his developing blindness.

II. Theories as to the cause of Milton's Blindness

It throws a vivid sidelight on the age in which Milton was living that Milton's enemies should publicly ascribe his blindness to a divine punishment for his transgressions—and that Milton found it necessary to deny such charges. And not only Milton but his biographers too.[6, 8] In an impassioned defence of himself Milton says :[4]

" . . . . I have accurately examined my conduct and scrutinised my soul, I call Thee, O ! God, the searcher of hearts to witness that I am not conscious either in the more early or in the later period of my life, of having committed any enormity, which might deservedly have marked me out as a fit object of such a callamitous visitation. But since my enemies boast that this affliction is only a retribution for the transgressions of my pen, I again invoke the Almighty to witness that I never at any time wrote anything which I did not think agreeable to truth, to justice and to piety. This was my persuasion then and I feel that same persuasion now. Nor was I ever prompted to such exertions by the influence of ambition, by the lust of lucre or of praise; it was only by the conviction of duty and the feeling of patriotism, a disinterested passion for the extension of civil and religious liberty."

* Philips says Milton's sight " had been decaying for about a dozen years." That puts back the onset of the trouble to the 31st year.

The cause of Milton's blindness was touched upon by two writers before the era of the ophthalmoscope. According to W. Lawrence[20] Milton suffered from " suffusion "; Mackenzie[21] held that the blindness was caused by " amaurosis " due to congestion or inflammation of the optic nerve secondary to a chronic disease of the digestive organs.

More recent writers have been more definite in their diagnosis, and four different suggestions have been advanced.

1. *Glaucoma.*—That Milton's blindness was due to glaucoma seems to be a popular view. This view first was advanced in 1879 by Alfred Stern[12] who says in his classical " Life of Milton," that he consulted a " distinguished ophthalmologist " on the nature of Milton's blindness and that according to this authority some of the features tended to a diagnosis of glaucoma, though others were against such a view. The reasons for this conception are not given. Glaucoma is also suggested by Hirschberg[13] in a footnote in his " History of Ophthalmology." Again no reasons are given. In an article on the subject in the " American Encyclopedia of Ophthalmology," T. H. Shastid[14] also suggests glaucoma—and again no reasons are advanced.

Apparently the main reason for the diagnosis of glaucoma is the fact mentioned by Milton in his letter to Philaras, that if he " looked at a lit candle, a kind of iris seemed to snatch it " from him. That this symptom led Hirschberg to the diagnosis of glaucoma is vouched for by Mutschmann[18] who declares that Hirschberg says so in a personal letter to him.

On the face of it there is much to support the view that glaucoma was responsible for Milton's blindness. Here is a progressive deterioration of sight extending over a period of years (somewhere between 8 to 12 years), associated with the phenomenon of iridescent rings around naked lights, and ultimately leading to complete blindness though the external appearances of the eyes are normal. But the diagnosis of glaucoma only holds good when the bare outlines are considered: the details do not fit in.

To begin with Milton was first troubled with his eyes at about the age of 35 according to his own account, at about the age of 31 according to the account of his nephew Philips. " Not very long " afterwards the " left part of the left eye " had failed. This certainly does not mean contraction of the nasal half of the field—nor can it mean the deterioration of vision following an acute attack of glaucoma, for such deterioration does not pick out particular parts of the fields. Besides it presupposes the glaucomatous process to have started very early in life if by the early thirties the sight of an eye could have been lost. By 43 Milton was totally blind. It is surely most unusual for chronic glaucoma —and it must have been chronic if it was glaucoma, as the " right

eye was failing perceptibly and gradually for a period of three years "—to lead to complete blindness by 43.

Thus if it is true that the chronic nature of the deterioration suggests glaucoma, the age of the victim is all against it. The objection to glaucoma is strongly fortified by an analysis of the symptoms as related by Milton. Chronic glaucoma is essentially a symptomless disease; it is true that vision deteriorates steadily but because central vision is unaffected till late the patient is not aware of it. Glaucomatous patients do not describe loss of field such as Milton describes The metamorphopsia of which Milton speaks, the micropsia, the illusion of objects moving, the " copious light glittering from shut eyes " are all symptoms that are never met with in glaucoma. The one outstanding symptom that is present in glaucoma is the seeing of iridescent rings, and because other symptoms are absent in glaucoma this symptom has usurped quite an unmerited place in the diagnosis of this disease. It certainly is not pathognomonic of glaucoma: it is met with in a number of other conditions, and in view of the fact that everything else points strongly against glaucoma, the presence of this symptom is quite insufficient evidence for such a diagnosis. It must also be remembered that when Milton was blind, he could still get " a certain little trifle of light " with his eyes in certain positions. The blindness from glaucoma is usually absolute.

Two further facts throw doubt on the diagnosis of glaucoma. One is our knowledge that Milton's eyesight was naturally weak. It may therefore be that this " natural weakness," if its nature could be ascertained, was ultimately the cause of the blindness. The other is the fact that on the one symptom on which one is tempted to diagnose glaucoma Milton appears to have been an unreliable witness. The iris around a candle of which Milton speaks may merely have been a play of colours, for it is said[19] that Milton only saw three colours in the rainbow. A play of colours around a light is decidedly more feeble evidence of glaucoma than a display of rainbow-colours.

2. *Detached Retina.*—In a short communication Dufour[15] of Lausanne advanced in 1909 the suggestion that Milton's blindness was due to detachment of the retina in both eyes. He bases his conclusions on an analysis of the letter to Philaras and on the fact that Milton's eyes were normal in their external appearances. Dufour stresses the illusions of deformity and mobility of objects looked at, the subjective sensations of light persisting after loss of sight, and the facts of " dimness of the visual field coming on first on the left side and then at the top; the sensation of steam before the eyes, showing the well-known picture of the narrowing of the visual field from above."

As to the cause of the detachment, Dufour holds that it was due to myopia, and in support of the theory that Milton was myopic he points out that Milton was an ardent student since childhood, and this would cause him to be myopic. As further proof is given the fact that though Milton travelled extensively, crossing " some of the most beautiful parts of the world " there is not in his work " one description, one line referring to the beauties of Nature." " Although in the seventeenth century no thought was given to the beauties of Nature, this attention to things heard, and indifference to things seen, appears to be the chief characteristic of the shortsighted."

In criticism of Dufour's view it must be said that though his analysis of the letter to Philaras makes out a strong case for detachment, his proof that Milton was myopic is not very convincing. Whether close work, even by poor candle light, can bring on myopia, is to say the least, very much in question, and to say that Milton was insusceptible to the charms of Nature is decidedly an overstatement. It is enough to mention the opinion of Walter Savage Landor on Milton's treatment of Nature: " If ever there was a poet who knew her well and described her in all her loveliness, it was Milton." That Milton should not have spoken of the natural beauty that he must have seen during his travels is quite satisfactorily explained by the fact that none of his contemporaries ever spoke of such things. Appreciation of the beauties of Nature is a remarkably recent acquirement—and only two hundred years ago Switzerland was regarded as a country full of hideous and inconvenient mountains. Milton may have been, and probably was, myopic, but the evidence advanced by Dufour is by no means convincing.

3. *Congenital Syphilis.*—In 1924 Saurat and Cabannes[16] advanced the suggestion that Milton suffered from congenital syphilis and that this affection was the cause of his blindness. They reject the suggestion of detachment, because they are not satisfied that Milton was myopic; indeed they hold that Milton was not the subject of myopia, basing their conclusion on the fact that Milton had good colour vision. In arriving at the conclusion as to Milton's good colour vision they base themselves on the work of Squires[19] whose opposite conclusion, viz., that Milton had poor colour vision they do not mention. They therefore argue against detachment on this hypothetical good colour vision. They also say that such of the symptoms described by Milton as are suggestive of detachment may also be met with in the last stages of chronic inflammatory processes of the retina and optic nerve. Excluding detachment (they say nothing against glaucoma) they therefore suggest neuro-retinitis, and hold that it was of syphilitic origin on the strength of some facts concerning Milton's health

and that of his family. The chief of these are the mortality in Milton's family, and the fact that Milton suffered from joint trouble (affecting the fingers and diagnosed as gout by his physicians). The further proof advanced is that Milton had feeble eyes in his childhood and that his mother's eyes were weak—and also that Milton's oval face suggests congenital syphilis to a physician.

This is remarkable evidence on which to diagnose congenital syphilis. Nothing at all is said of the fact that Milton was one of six children—of whom three survived—and that Milton was the third, the two others who survived being the second and the sixth. The survival of the second, third and sixth does not suggest syphilis, and the death of three out of six children is nothing unusual in mediaeval chronicles and certainly poor evidence for the diagnosis of syphilis. How the fact that Milton's mother had weak eyes is a support for this theory is by no means obvious. Supposedly she was suffering from syphilitic inflammation of the eyes and we are to believe that it was for this that she took to wearing glasses. Such a belief certainly lacks nothing in boldness. The other evidence as to Milton's congenital syphilis is the mortality among his children and grandchildren, and also the fact that his brother lost three children in their childhood. Congenital syphilis with such widespread influences persisting for generations is indeed a remarkable phenomenon. The immediate progeny of a congenital syphilitic usually show no strain of syphilis: in Milton's case his grandchildren were still dying from it. The exceptional qualities of Milton were apparently not confined to his poetical achievements: the congenital syphilis ascribed to him by Saurat and Cabannes is even more exceptional. One of Milton's daughters and his first two wives died in childbed. This apparently was also due to the congenital syphilis of Milton, for these deaths are recorded in the list of catastrophes for which congenital syphilis is the explanation.

The symptoms that Milton describes during the onset of his blindness are said to fall in with the suggestion of a lesion of congenital syphilitic origin. In a world where everything is possible, this of course cannot be ruled out, though most oculists expect a different type of clinical picture for congenital syphilis. Interstitial keratitis and iritis are, with their obvious external lesions, more common conditions in congenital syphilis than lesions which could give rise to obscuration of definite parts of the visual fields and distortion in the appearances of objects. It must be admitted that some—and only some—of the symptoms described by Milton can be explained on the hypothesis of disseminated choroiditis. But any other chronic lesion would do as well—hydatid cysts of vitreous among others. The fact that Milton's eyes were " weak " since childhood is important evidence, but in itself no evidence at all that he suffered from congenital syphilis.

Milton's oval face we are also told suggests congenital syphilis. Milton's face must have possessed highly suggestive properties: to his fellow students it suggested the nick-name of the Lady of Christ College,[8] to a contemporary lampooner of his it suggested " a horrid monster "; to a modern critic[17, 18] who thinks that Milton was an albino it suggests albinism. And no doubt it has suggested other things to other critics besides Saurat and Cabannes.

Milton's family history is definitely against congenital syphilis and the nature of Milton's eye trouble does not contain one shred of evidence in favour of such a view. Congenital syphilis as the cause of Milton's blindness is not only a far-fetched theory, but one that flies in the face of all established facts. A rather less painfully laboured case could be made out for acquired syphilis—less painfully laboured but no less obviously impossible.

4. *Albinism.*—The theory that Milton was an albino is really outside the scope of the present article, for the author of that theory —H. Mutschmann,[17, 18] a professor of literature and not a medical man—does not claim that albinism was the cause of Milton's blindness. He accepts Hirschberg's view that glaucoma was responsible for the final catastrophe.

Mutschmann's views are, however, of interest as they touch upon the nature of the " natural weakness " of Milton's eyes. It must be added that Mutschmann has no interest in this question apart from the fact that it is part and parcel of a theory of his that Milton was a physical and moral degenerate, and that all his life and work was only a striving after power, that he was a potential criminal and kept out of criminal activities by his cowardice, and so on and so forth. It is not surprising that Mutschmann should think that some of the accepted views concerning Milton are " impertinent concoctions " (*eine freche Erfindung*). Those who are interested in the remarkable *tour-de-force* by which all this is " proved " can refer to the voluminous efforts of their author—both in German and in English.

That Milton was an albino is argued on two lines: evidence is advanced from the biography by Aubrey and this is supported by internal evidence from Milton's writings. Milton, according to Aubrey had brown hair: a marginal note makes the brown into abrown; furthermore Milton was " exceeding faire." The fact that Milton's hair was " abrown " means to Mutschmann that it was not brown, that is white. White hair being proved the " exceedingly faire " complexion proves albinism. That his eyes according to the same biographer (who knew Milton) were a " darke gray " does not upset Mutschmann very much. That Milton had white hair is further proved to Mutschmann's satisfaction by the reddish tinge of his hair seen in some portraits.

The fact that the earliest life of Milton extant, the one ascribed

to Nathan Paget, speaks of Milton as having a ruddy complexion and light brown hair is not even mentioned by Mutschmann. On the " evidence " that Mutschmann has collected he has reared a colossal superstructure of albinism—and as if it were a natural corollary—of physical and moral degeneracy. Milton's albinism is further proved by reference to his writings from which Mutschmann is satisfied that Milton among other phobias had photophobia and was a nyctalope. On the literary merits of this criticism Mutschmann can be left to the recognised authorities on Milton. He has been dealt with faithfully by Stern, the veteran Milton scholar of Germany, amongst others. Here it is only necessary to point out that there is no disproving Mutschmann's evidence as to Milton's albinism, for there is no evidence to consider. What he says about Milton's appearance are not facts but distortions.

Mutschmann has drawn up an elaborate chronology of Milton's works on the hypothesis that Milton betrays his photophobia in his writings up till 1644. In that year he got his " first attack of glaucoma " and that " would have the effect of suddenly reducing the sensibility of the retina so considerably, as to cause the disappearance of existing photophobia without however completely destroying the sight."

Glaucoma which acts by " suddenly reducing the sensibility of the retina " cannot but be acute glaucoma. And unless Milton had acute glaucoma in both eyes in 1644 (and there is no evidence of involvement of the right eye till much later) we must believe that it was only the left eye that was albinotic. Albinism confined to one eye is a decidedly interesting subject.

5. *The " natural weakness " of Milton's eyes and his blindness.*—From what has been said it is clear that the most important document available, the letter to Philaras, does not in itself solve the problem of Milton's blindness. The two views deserving attention that have been advanced on the strength of this letter are that the blindness was due to glaucoma, or to detached retina. The reasons for rejecting glaucoma have been advanced : glaucoma only explains the broad outlines but not the details that we have, and some of these details go most decidedly against glaucoma.

Against detachment the arguments are that no satisfactory proof is advanced by the exponent of this theory that Milton was myopic and that he ignores the question of iridescent rings of which Milton speaks. As to these rings it has already been pointed out that they do not by any means always argue for glaucoma, and certainly cannot be taken to mean glaucoma when as in this case all the other symptoms and available evidence are against glaucoma. The symptoms described by Milton may therefore be taken to mean detachment if the pre-existing condition of myopia can be proved. The graphic description of obscuration of the left side of the left

eye and ultimately complete failure of that eye, strongly suggests detachment leading to disorganisation of the eye, particularly if taken together with the description of the failure later on of the right eye. " I observed some months before my sight was wholly gone that objects I looked at, without myself moving seemed all to swim." Perception of light and some functioning retina remained, as it almost invariably does in such cases: " The darkness which is perpetually before me by night as well as by day, seems always nearer to a whitish than to a blackish, and such that when the eye rolls itself, there is admitted as through a small chink, a certain little trifle of light."

It is, of course, possible that the right eye was suffering from a progressive inflammatory choroido-retinitis which would explain most of the symptoms, though hardly the swimming of objects looked at. The difficulty of detachment as a diagnosis in the case of the right eye is that it was " failing perceptibly and gradually through a period of three years." Detachment is usually abrupt in onset though the deterioration it causes is progressive for a considerable time before complete blindness sets in. In view of the fact that the swimming of objects only set in some months before the sight was wholly gone, and at the time when " inverterate mists " made their appearance, it is not unlikely that detachment was the culmination of active myopic degeneration which had been going on for some years.

Abrupt detachment in the left and detachment in the right following on progressive myopic changes in that eye seems to be a likely diagnosis on the strength of Milton's letter, and not an unlikely diagnosis from what we know of myopia.

It therefore remains to consider what evidence there is for the view that Milton was myopic, and in attempting to establish myopia it is not at all necessary to presuppose a high degree of myopia, for detachment is as prone—if not more so—to attack the medium myope as the high myope. This brings us back to the question of the natural weakness of his eyes to which Milton testifies. Was this natural weakness myopia?

It is quite impossible on the available evidence to come to a definite conclusion, but there is a strong presumptive evidence that it was indeed myopia which was the cause of his weak eyes, and led ultimately to his blindness. The evidence comes from varying sources:

(1) *Milton's heredity:*

Milton's mother " had very weak eies and used spectacles presently after she was 30 years old." We have no nearer definition of her defect than this statement. It could not have been astigmatism for which she took to glasses, for an astigmatic

correction was first made use of by Airy in 1827. Thirty is too young an age for presbyopia, so that the choice is left between hypermetropia and myopia, and of these myopia is much the more likely condition, for hypermetropia very rarely gives rise to such a severe visual defect as is implied in Aubrey's statement. Of course it is possible that the " weak eies " were not visually defective but subject to chronic conjunctivitis or blepharitis, but it is unlikely that glasses were worn for such conditions at that time. " Weak eies " in all probability means a high degree of myopia.

There is also a strong probability that Milton's father was a myope. Aubrey says that he could read without glasses at 84. Only a myope can do that, though the myopia need not be of the axial type. It is just possible that this myopia of Milton's father at 84 was not of the axial type but due to incipient cataract. But again axial myopia is the more likely.

Thus if Milton's father and mother were both myopic, it is not at all unlikely that Milton too was a myope, for the strong hereditary tendency in myopia is well known.

(2) *Milton's habits:*

Milton tells us that his eyes were " naturally weak." Whether this weakness means in Milton's case what it meant in his mother's we do not know, for there is no record of Milton taking to glasses. We know he was an ardent student from his early days, with brilliant scholastic attainments; he shunned society and was not popular amongst his gay fellow-students. He was what is colloquially known as a " bookworm," exhibiting all the characteristics popularly ascribed to the spectacled student. His habits are in agreement with the suggestion that he was of myopic stock and that the weakness of his eyes was indeed myopia.

(3) *The internal evidence from Milton's writings:*

The support based on the internal evidence of Milton's writings comes from a disinterested source. V. P. Squires[19] in analysing Milton's treatment of Nature shows—and this is generally accepted —that Milton was none too good an observer, and the following lengthy extract gives the conclusions reached on the careful analysis he makes of Milton's references to Nature.

" This lack of intimate knowledge sometimes caused him to make mistakes as in his allusion to the pine tree in ' Paradise Regained,' IV, 416-17, and in ' Lycidas ' where he groups together flowers of different seasons which could not possibly be found in bloom at the same time. Another fact which forces us to this conclusion is his lack of fine distinctions in colours. He mentions green and blue, but never refers to their various shades. In Wordsworth, for instance, we find not only green, but olive

green, pear green, dark green, etc., not only blue, but pale blue, dark blue, sable blue, black blue, etc.

" These failures to be exact may possibly have been occasioned by near sightedness. We do not have a record that Milton was thus afflicted, but we do know that he had some trouble with his eyes, that he strained them by overstudy in his youth, and that they were a continual source of anxiety to him. Excessive study in youth nowadays usually tends to myopia,* and so it is not unreasonable to suppose that this was the nature of Milton's ailment. The effect of myopic vision is to render outlines indistinct, and hence to make impressions indefinite. It is impossible to distinguish one sort of bird from another or one sort of tree from another, altho' the observer is fully aware that a bird or a tree is before him. This seems to have been precisely the case with Milton. He mentions definite birds very rarely, except the nightingale and this is always spoken of in connection with its singing at night, when of course it could not be *seen*. The failure to make nice distinctions in colour also could be explained by the same theory, as could the fact that he saw three colours in the rainbow.† Another bit of evidence is his fondness for bright glittering objects."

This is the evidence in favour of myopia, and as far as it goes it certainly does not contradict the suggestion that Milton's blindness was ultimately due to complications of myopia. On the contrary it lends some support to such a view.

One piece of autobiographical detail must be considered as going against the conclusion that Milton was a myope, and this is Milton's statement supported by his first biographer that he wore a sword and had skill in using it. It must, however, be remembered that these statements were written in defence against attacks on Milton's character, and to wear a sword and use it was a necessary equipment of a gentleman's character in those days. Besides, a myope of medium degree, as Milton probably was, might very well be able to use a sword, for sword play after all takes place at fairly close quarters, and should not be impossible to one not very highly myopic. But Milton was certainly no man of war. He did not serve in the army during the whole of the civil war, though he was an ardent Parliamentarian, being satisfied with more bookish exploits. His later years were of necessity years of passive activity, but his earlier years too give the picture of a quiet man.

But all these speculations are only about the man and not about the creator of the polysyllabic splendour of " Paradise Lost." To

* This is questionable.

† I have not been able to trace the source from which this statement is made.

attempt to trace the influence of Milton's eyesight and of his blindness on his poetical achievements is an idle task, for Milton, whose soul was like a star that dwelt apart, is like Shakespeare, free—though others abide our question.

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A REPORT ON THE USE OF VITAMIN "A" IN CONJUNCTION WITH ULTRA-VIOLET LIGHT IN OPHTHALMIC DISEASES

BY

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ANY remedy or therapeutic measure which is acclaimed, especially in the daily press, as a panacea, should be approached with suspicion. For this reason much harm has been done to the legitimate use of ultra-violet light in the minds of reasonable people by those advocates who have proclaimed it as the cure for any and every bodily ailment.

Such a condition of affairs is largely due to the fact that for some years this form of treatment has been in the hands of unqualified persons; but, if it is recognised that it has, in common with any other specialised form of treatment, a well-defined and limited application, it will then be given its proper place among modern methods of ophthalmic treatment.

That it has a definite application may be seen from the following report on cases which have been attended in the ultra-violet light clinic of the Central London Ophthalmic Hospital.

This clinic has now been working for three years and records of 181 cases are available for analysis. The clinic was inaugurated by Miss Ida Mann and was under her supervision during the first year when 64 cases were treated. I am indebted to her for permission to use the records she then obtained.

The lamp used is a carbon-arc which gives a steady output of ultra-violet rays. In the case of mercury vapour lamps the output diminishes as the lamp gets older, so that without frequent control, exposures necessitating some delay, the dosage cannot be accurately measured by the clock.

As a preliminary, those cases suffering from arteriosclerosis, Bright's disease, cardio-vascular lesions, glycosuria, menorrhagia, and pulmonary tuberculosis, are excluded as such are very frequently harmed by ultra-violet light.

General exposures of increasing duration are given tri-weekly in serial rotation so that the same skin surface is not exposed on two successive visits. Signs of increased temperamental irritability, insomnia, persistent loss of weight, nausea, anorexia, and a raised temperature 24 hours after exposure, indicate an over dosage, and should any of these occur the treatment is stopped.

All cases treated in this clinic are examined at least once a fortnight when a note is made of the condition and progress of the disease.

At the end of two years it was discovered that if improvement were to be obtained from this treatment it was noticeable at the end of 12·9 exposures (this figure agrees with that of 12·5 found by an independent observer working under similar conditions el ewhere and without previous collaboration), so patients are now given " courses " of 15 exposures. Since after a large number of exposures the skin becomes tolerant to the rays, and since too many or too prolonged exposures result in debility and depression, these " courses " are punctuated by a month's interval when no light treatment is given.

As a result of this regime there has been a saving of the patient's time and the hospital's purse, and a larger number of patients have been accommodated in the clinic—in a word, greater efficiency.

During the last year Vitamin A has been given to the patients concurrently with the ultra-violet light. This vitamin has been

shown by Green and Mellanby to have anti-infective properties, and the tables below show the comparative results obtained with and without this addition to the treatment. The action of the vitamin is more pronounced when given in conjunction with an oil or in an oily vehicle.

The patients all state that they feel better "in themselves" for the treatment—eat better, sleep better, and in most cases put on weight. Very few cases fail to attend regularly, showing that they appreciate the benefit derived.

The best results have been obtained in phlyctenular ophthalmia, hordeola, episcleritis, corneal ulcers and tuberculous iritis. In other conditions the results are good but not, to my mind, convincing: that is to say, though improvement has taken place it is difficult to determine whether it has been due to the ultra-violet light or would have occurred as a result of the ordinary routine treatment employed at the same time.

It will be noticed that the conditions mentioned above as being benefited by the rays occur superficially or in the anterior segment of the eye, while those more deeply seated, such as vitreous opacities, choroiditis, optic neuritis, etc., do not seem to improve to an extent which justifies the time given up to the treatment by the patient.

The more cases that are treated with ultra-violet light, the more surely can those cases which will derive benefit be selected, the more efficiently will a light clinic be administered, and the more will the true value of this treatment be appreciated.

TABLE I.

COMPARISON BETWEEN 53 CASES WHICH RECEIVED VITAMIN "A" AND 86 CASES WITHOUT THIS ADDITION.

a With Vitamin "A"

	No. of Cases.	No. of Exposures.	Average Exposures.	Point at which improvement first recorded. No. of Exposures.	Point at which improvement first recorded. Average Exposures.
Cured -	38	587	15·4	233	6·1
Relieved -	15	255	17·0	87	5·8
	53	842	16·0	320	6·0

b Without Vitamin "A"

Cured -	40	1,060	26.5	485	12·5
Relieved -	46	1,306	28.8	545	11·3
	86	2,366	27·1	1,030	11·8

TABLE II.

COMPARISON OF SELECTED DISEASES.

Disease.	*With Vitamin "A"*				*Without Vitamin "A"*			
	CURED		RELIEVED		CURED		RELIEVED	
	Cases	Exposures	Cases	Exposures	Cases	Exposures	Cases	Exposures
Conjunctivitis-	3	41	1	15	2	111	1	12
Corneal Ulcer-	5	69	2	24	10	140	7	114
Episcleritis -	3	58	—	—	4	92	2	51
Interstitial Keratitis -	2	37	3	61	2	53	8	194
Phlyctenular Ophthalmia -	11	125	3	39	10	256	6	106
	A total of 33 cases had 469 exposures.				A total of 52 cases had 1,131 exposures.			

HAEMANGEIOMA OF THE ORBIT

BY

THOMAS H. CRESSWELL, D.O.Oxon. and
W. ARTHUR BRIGGS, D.O.M.S.

LINCOLN

MRS. C., aged 26 years, a patient of Dr. Armour, of Woodhall Spa, was seen by us on January 24, 1930.

She complained that two years ago she had noticed some defect in vision in her left eye and that this had gradually become worse. Six months ago she noticed that this eye had become somewhat prominent and that the prominence had gradually increased to its present state. She made no further complaint at all.

On examination, it was seen that this left eye was proptosed to the extent of about 0.75 cm. The movements were full in every direction and no diplopia could be elicited. There were about five dioptres of hypermetropia and vision with a plus 5.5 D.sph. was 6/12. There was a mild, though quite definite degree of papilloedema as shown by fullness of the vessels and blurring of the disc margins,

but gross signs, such as pulsation of vessels, haemorrhages, etc , were absent. There was no bruit and depression of the head produced no further proptosis. The effect of compression of the jugular vein was not tried. It may further be added that the globe could not be pushed back into the orbit and that there were no signs such as tachycardia, tremor or other typical signs of exophthalmic goitre. It was, however, observed that the patient had two well-marked scars in the region of the orbit, the results of an injury by a branch of a tree when she was aged nine. A diagnosis of orbital new growth was made and this diagnosis was supported by Mr. Malcolm Hepburn.

Operation

A curved incision, 1½ inches long was made in the outer side of the conjunctiva, close to the fornix. The external rectus muscle was exposed and divided, the severed ends being secured by sutures. The external canthus was then divided and a sagittal section made in the muscle cone. The optic nerve was found free for about ½ inch

behind the globe when its track was lost, being apparently diverted by a mass of abnormal tissue. One of us attempted to pass the index finger behind the growth and though it did not seem as if much force were being employed, the growth, compressed between the finger and the outer wall of the orbit, was suddenly delivered, jumping out as much as a foot from the wound. The sensation which accompanied this movement was that of flipping a fresh cherry stone between the finger and the thumb. When freshly delivered, the tumour was the size of a medium-sized strawberry, spongy, heart shaped, liver coloured and enclosed in a glistening capsule ; this latter appeared incomplete only at the bleeding points where the in and out going vessels had been severed.

Being uncertain as to the nature of the growth and realizing that the contents of the orbit had probably sustained considerable damage, it was decided to complete the sub-total exenteration of the orbit.

Pathologist's Report

Dr. E. S. Winter, pathologist to the County Hospital, Lincoln, reports as follows:—

The specimen, which is heart shaped, liver coloured and surrounded by a thin glistening capsule, measures rather over 2.5 cms. in length, 2.5 cms. in breadth and 1.5 cms. at its thickest part. On removing a small piece for sectioning, the contents seemed to consist of innumerable small spaces, separated by well-formed tissue and containing blood. Microscopically, the section shows irregular communicating spaces with walls lined with endothelial cells and filled with blood corpuscles. The specimen is a haemangeioma.

ABSTRACTS

I.—RETINAL DETACHMENT

Rubbrecht, R. (Bruges).—The closure of holes in the retina. (L'Obturation des Déchirures de la Rétine). *Arch. d'Ophtal.*, March, 1930.

In this paper **Rubbrecht** deals only with operative technique, leaving out of consideration the rôle of retinal tears in the pathogenesis of detachment. After a tribute to Gonin as the pioneer of the treatment under discussion, he alludes to the difficulty often experienced in finding the hole in the detached retina; more than one examination may be necessary. The tear may be situated at the extreme periphery of the ophthalmoscopic field; it may be in a part of the retina which has become re-applied and distant from the actual detachment; it may be hidden by a fold of the retina and only discoverable when the patient has been recumbent for several days. Having found the hole it is necessary to localise it as exactly as possible, in order that the site may be indicated as nearly as is feasible on the external surface of the sclera. For this there are two essentials:—(1) to determine the meridian in which the tear lies, and (2) to calculate the distance between the lesion and the corneal limbus; or, as the author suggests, " to fix the longitude and latitude." Rubbrecht's plan is as follows:—The patient is placed in a horizontal posture and looks directly upwards; the examination is made of the upright image.

In Fig. 1 the retinal tear is at D: the observer at A sees it projected on the edge of the pupil; this being dilated to its maximum,

the image of the tear appears to be a very short distance from the limbus, and the corresponding point on the limbus is marked with Chinese ink. The observer then shifts his position to B and sees the image of the tear projected on the pupillary border directly opposite that already noted. A second mark is made at the limbus at

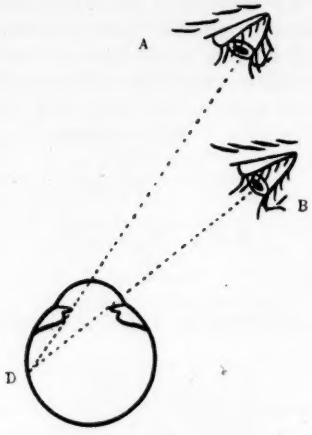

FIG. 1.

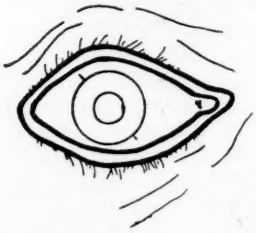

FIG. 2.

this point, and thus we have, indicated very accurately, a diameter of the cornea which forms part of the circle of longitude in which the retinal hole lies (Fig. 2).

The next essential is to determine the meridian in which the incision is to lie. The simplest plan is to calculate in disc-diameters the distance between the tear and the ora serrata. This latter is not always visible and then it suffices to take the extreme point visible with the ophthalmoscope. This examination should be made with the patient lying down. The calculation should be made with all possible care, but absolute exactness is neither

possible nor essential. As will be seen later, in the method here described, the intervention is not on one point but on a certain extent of a meridian, and this lessens notably the risk of error. Having determined the distance of the retinal tear from the ora serrata it is necessary to ascertain its distance from the limbus. From the limbus to the ora serrata is reckoned as 8 mm. If, for example, the tear is found to be 3 disc-diameters from the ora serrata it should be 8+4·5 mm. = 12 mm. from the limbus. Rubbrecht states emphatically that these examinations and measurements are of prime importance; if necessary several days should be devoted to them.

Thorough local anaesthesia having been induced by novocaine and adrenaline, the steps of the operation follow in definite order.

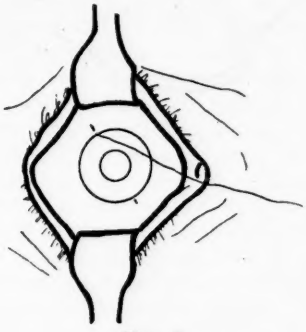

FIG. 3.

A thread is inserted at the limbal ink-mark farthest from the site of the incision (Fig. 3). This thread is knotted at the end. Resection of the external or internal rectus is then performed: this resection, which is purely temporary, is helpful as it enables the assistant to grip the globe firmly by the cut tendon and to rotate it sufficiently to expose the sclera for incision. Stitches are passed through the two parts of the muscle and these are brought together accurately immediately after the operation. The conjunctiva, and episcleral tissue, are incised parallel to the limbus on the side of the detachment. The area of sclerotic laid bare should measure at least 1 cm. antero-posteriorly. By means of a retractor the posterior edge of the conjunctival wound is pulled backwards and a haemostatic applied carefully to the exposed surface of the sclera to obviate any interference with the further procedure, by bleeding. The thread already inserted in the episcleral tissue and retained by a knot now becomes a directing thread (" le fil conducteur "). It is brought across the cornea and passed through the second limbal

ink-mark (Fig. 4) and carried onwards in the same line over the denuded sclera. This indicates the meridian in which the retinal tear lies, and an ink-mark is drawn on the sclera along the course of the thread. The directing thread is now removed. Assuming now that the site of the tear has been measured as 14 mm. from the limbus, the incision is begun at a point 12mm. from the limbus and continued along the meridian shown by the ink line to a point 16 or 17 mm. from the limbus, thus having a length of 4 to 5 mm. The incision is made with a Graefe knife very cautiously, layer by

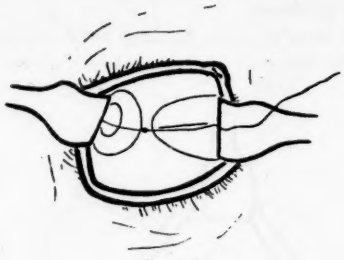

FIG. 4.

layer, to avoid sudden penetration of the vitreous. The choroid is incised and the subretinal fluid allowed to escape. The galvano-cautery at a bright red heat is passed rapidly in the whole length of the incision. Rubbrecht prefers a sickle-shaped terminal: he does not attempt to plunge the terminal into the vitreous, and indeed he disapproves of such a procedure. The application of the cautery occupies only a few seconds. The resected muscle and the conjunctiva having been sewn up, a bandage is applied over both eyes for 24 hours. The patient is kept in bed for 8 days; atropine is applied to the operated eye once daily.

J. B. LAWFORD.

II.—REFRACTION

(1) Ferree, C. E., Rand, G. and Monroe, M. M.—Errors of refraction, age and sex in relation to the size of the form field and preliminary data for a diagnostic scale. *Bulletin of the Johns Hopkins Hospital*, Vol. XLV, p. 295, 1929.

(1) **Ferree** and **Rand**, whose careful work on standardising the conditions for perimetry is well known, take up in the present article the variations in the form field with different individuals. The importance of such a study (in which they had the help of

Monroe) is obvious, if perimetry is to be used as a means of diagnosis by revealing early changes in the field. " Previous writers on this subject seem to have been content to give the breadth of field in the principal meridians for the average case, or at most only a small range of variation on either side. The knowledge of what is an average field obviously contributes but little to the diagnosis of cases for which the help of perimetry is most needed. If a pathological condition is to be diagnosed in its incipiency, the border line between normal and pathological conditions must be located within reasonably narrow limits. For example, a given field may be much smaller than the average and still fall within the range of normal variation."

The present study has been carried out to determine the range of variation of the form field for non-pathological eyes for a stimulus subtending a visual angle of one degree. The cases studied, two hundred in number, were sampled to include as many as possible of the outstanding variables which are not pathological. Errors of refraction were found to be an important factor influencing the variability of results from individual to individual; in general the emmetropes and hypermetropes have wider fields than the myopes. Presbyopia, too, has an effect; under 40, age has no influence on the field, but the onset of presbyopia tends to make the field narrower. Sex would appear not to be a factor of sufficient importance to be taken into account in diagnosis. The authors have established a scale showing the range of variation of field under the influence of these variables; and also showing the distribution or frequency of occurrence of the different sizes of field in a typical non-pathological group. " Such a scale provides a means of placing each new case in the class or group to which it belongs, rated with regard to the distribution of normal cases, *i.e.*, it constitutes a scale for use in the separation of pathological from non-pathological cases."

ARNOLD SORSBY.

(2) **Tron, Eugen (Leningrad).—Statistical research on variations in refraction. (Variationsstatistische Untersuchungen ueber Refraction).** *Arch. f. Ophthal.*, Vol. CXXII, p. 1.

(2) **Tron** has studied the measurements of a series of 200 eyes, showing a refraction varying from +7D. to -25D., by means of the ophthalmophakometer, and his investigations on the variability of the dioptric elements of the eye show that all these elements (depth of the anterior chamber, thickness of the lens, radii of the refracting surfaces, the refractive power of the cornea, lens, and the whole eye) form a variational curve that exhibits a substantial agreement with the normal binominal curve.

The sole exception to this is the variational curve for the axial length, which is characterised by its great asymmetry and considerable height. These features disappear when all cases of myopia of -6D. and over are excluded from the series, and the axial length then shows a curve corresponding with the binominal.

The marked asymmetry and height of the curve for the axial length of the entire series of cases is due to the fact that it is to a certain extent made up of two curves: a normal variational curve for axial lengths of eyes between +7D. and -6D., and a second curve for cases of high myopia which takes an irregular course and is added on to the first.

The fact that in eyes between +7D. and -6D. the axial lengths form a normal variational curve indicates that the different axial lengths of these eyes are merely links in a normal variational series. Hence it is impossible to accept the theory that an arrest of development is the cause of a short axial length in hypermetropia, or that the lengthening of the axis in myopia is due to close work and pathological factors.

The refractive power of the lens is extremely variable, this variability, though little importance has been attached to it, being no less than that of the cornea. Hence any determination of the axial length in the living eye that only takes account of the corneal refraction and employs for the lens refraction the number for the schematic eye, thereby neglecting the normal variability of the lens refraction, is incorrect, as the author demonstrates in a table of examples.

In finding the relation between refraction and the different dioptric elements only the error of the particular ophthalmometric method employed was formerly considered, and the final conclusions were based on a comparison of the average values. The variational statistics, however, show that the average value of a variational series empirically ascertained differs more or less from the true average of that series because of the existence of the average error.

The statistical and variational study of this series of eyes, taking into consideration the average error for every average value, brought out the fact that as regards the elements of the optical apparatus of the eye, with the exception of the axial length, no real differences can be found between emmetropia on the one hand and hypermetropia and myopia on the other. In the case of the axial length there are definite differences between the different types of refraction, and yet the variational limits in the axial length overlap to some extent in these types. Hence one can conclude that the axial length in eyes of different refraction may be the same.

The calculation of the correlation-coefficient between refraction

and axial length for the entire series as well as for the different types of refraction brings out figures that are only slightly over 0·5. The fact that there is a positive correlation between refraction and axial length proves that the absolute length of the axis has by itself no definitive importance for the refraction. The analysis of the correlation between refraction and axial length on the one hand and the refractive power of the eye on the other, and the existence of only slight, and even negative, correlation between axial length and refractive power of the eye show that in the majority of cases the refraction depends neither on the absolute axial length nor on the refractive power of the eye, but on a combination of both these factors.

THOS. SNOWBALL.

III.—GENERAL MEDICINE

(1) **Friedenwald, H. and Friedenwald, J. S. (Baltimore).—The retinal blood vessels in hyperthyroidism and arteriosclerosis.** *Bull. Johns Hopkins Hospital,* Vol. XLV, p. 232, 1929.

(1) **H. and J. S. Friedenwald** review our present knowledge of retinal arteriosclerosis and emphasise the difficulties and problems under consideration. They point out that even the significance of such marked features as venous compression and altered light reflex on the arteries is still a matter of controversy, and the exact mechanism of such changes is a very much disputed problem. Their own contribution consists of an attempted classification of the retinal arteriosclerotic changes in relation to the general condition—a classification based on the examination of two thousand cases. Their grouping is as follows:

(1) *Hypertension without retinal arteriosclerosis.*—Hypertension from any cause—including essential hyperpiesis—may exist without retinal sclerosis, though venous compression is seen more frequently in such patients than in normal individuals. In this connection it is of interest to note that the hypertension developing with old age is not associated in the early stages with changes indicative of retinal sclerosis.

(2) *Retinal arteriolar sclerosis and albuminuric retinitis.*—The characteristic features of retinal arteriolar sclerosis are localised variations in the calibre, irregular tortuosity, and visibility of the walls of the terminal arterioles. These vessels can be studied better at the macula than at the periphery of the fundus. " In regard to the

tortuosity of these vessels a distinction must be made between the irregular tortuosity characteristic of vascular disease and the smooth sinuous tortuosity which is often seen in congested fundi in fever, eyestrain, etc." In addition to these arteriolar changes, the larger arteries usually show the characteristic features of sclerosis. The significance of this group lies in the fact that it must be regarded as the precursor of albuminuric retinitis, for all cases of this type that were followed up, ended as renal retinitis. Furthermore all cases of renal retinitis show this arteriolar sclerosis, as can be seen even in those cases where oedema obscures the macular region, for in these cases the peripheral arterioles still show this change. Clinically, marked hypertension is present, and sooner or later a rapidly progressing chronic nephritis becomes manifest. " Indeed all the well-known facts regarding age incidence, clinical associations and prognostic significance may be transferred bodily from the cases of albuminuric retinitis to the somewhat larger group of retinal arteriolar sclerosis."

(3) *Retinal arteriosclerosis with hypertension.*—Typically this occurs in old people with arteriosclerosis of the slow progressive type associated with hypertension. A characteristic feature is the reduction of the calibre of the whole arterial tree. " The relation of hypertension to the calibre is so close that the blood pressure can usually be estimated within quite narrow limits from the consideration of this sign alone. As a rough measure it may be said that when the arteries are reduced to approximately one half of their normal calibre, the diastolic blood pressure will be found 110 mm. or more." An important exception is the group of cases in which this retinal picture is associated with a low or normal blood pressure. In such cases the blood pressure has been lowered by some intercurrent disease, and cerebral thrombosis is a grave danger.

Apart from the highly significant constriction of the lumen, the other changes of arteriosclerosis are of course also present, though the larger arteries rather than the terminal arterioles tend to be implicated. Any retinal changes present (haemorrhages, exudates) are scattered over the fundus and not concentrated at the macula. The authors warn against regarding constricted retinal arteries as a physiological change of senility.

(4) *Retinal arteriosclerosis without hypertension.*—All the characteristics of retinal sclerosis are present, except that the arteries are full and tortuous. With such a retinal picture, not only is there no evidence of hypertension, but there is also no evidence of pre-existing hypertension that had been lowered by intercurrent disease. However, when this condition occurs in young people it is frequently only an intermediate stage going on to hypertension.

(5) *Syphilitic arteriosclerosis.*—The authors hold that when a

single branch shows evidence of arteriosclerosis, this points strongly to the lesion being syphilitic in origin.

(6) *Arteriosclerosis resulting from local causes.*—Arteriosclerosis is frequently the result of a purely local lesion, such as retinitis pigmentosa.

The paper is well worth reading in full.

ARNOLD SORSBY.

(2) **Igersheimer, J. (Frankfurt).—Affection of the optic nerve in malignant (vascular) sclerosis.** ("**Sehnervenerkrankung bei maligner sklerose.**") *Zeitschr. f. Augenheilk.*, Bd. CXIX, p. 47, 1929.

(2) **Igersheimer** draws attention to isolated affections of the optic nerve quite apart from neuroretinitis, as not uncommon complications of arteriosclerosis. A full account is given of the clinical and post-mortem findings in a case of his concerning a man aged 49, who suddenly developed disturbances of vision found to be due to optic neuritis. Apart from an old history of apparently cured syphilis, general examination showed nothing of importance. Within less than a month the patient was completely blind, and a few days afterwards he died from cerebral haemorrhage. The diagnosis on the strength of the post-mortem examination (which revealed no syphilitic lesions) was malignant arteriosclerosis with hypertension.

The retinae were hardly affected, but both optic nerves showed extensive degeneration, most marked near the disc; there was but little evidence of inflammatory changes, and no possibility of the optic nerve atrophy being the result of these. In the author's opinion the atrophy of the nerve was the result of circulatory disturbances, possibly aided by arterial spasm.

ARNOLD SORSBY.

(3) **Lange, Fritz (Munich).—Haemorrhages in the fundi in hypertension.** *Arch. of Ophthal.*, November, 1929.

(3) **Lange's** paper opens with an interesting example of how a false conclusion can be made from statistical observations. In 75 per cent. of cases from the ophthalmic department, the haemorrhages were in the region of the fovea, while in cases from the medical clinic, the haemorrhages occurred only in the periphery. The author points out the obvious reason, *viz.*, that macular haemorrhages cause a disturbance of vision which sends the patients to the ophthalmic department, but he also points out how easily a false conclusion could be formed about the area of predilection of haemorrhages if material from only one clinic were considered. The cases are divided into three groups: (*a*) Pure

hypertension, where the systolic and diastolic pressures are high, without there being any involvement of the kidneys or sclerosis of the peripheral arteries. (*b*) Pure arteriosclerosis, where the large and small arteries are sclerosed, but the blood pressure is normal or subnormal. (*c*) Mixed forms, where there is arteriosclerosis with high blood pressure. Seven different clinical methods are given for differentiating the first two groups. One of these is to place a cuff round one arm and observe the blood pressure, cuffs are then placed round the other three limbs and inflated at the same time. In hypertension there is a great rise in blood pressure while in arteriosclerosis there is almost no change. Of the 60 cases examined, 57 had a systolic pressure over 160 mm. Hg. Fifteen of these were pure hyperpietics. The main change in the arteries was unevenness of calibre, and in two cases the contractions disappeared after some weeks, while in the remainder they were permanent. These unevennesses were usually present when the blood pressure exceeded 200 mm. Hg and were absent when it was under 180 mm. Hg. There was no instance of retinal haemorrhage recurring in a case of pure arteriosclerosis without hypertension. In cases of haemorrhage of other origin, such as syphilitic or diabetic, high blood pressure was present with few exceptions. The majority of the haemorrhages originate from the capillaries.

F. A. W.-N.

IV.—BACTERIOLOGY

(1) **Morax (Paris).—Le bacterium granulosis et le trachome.** *Rev. Internat. du Trachome,* January, 1930.

(1) The four Indians from whose conjunctiva Noguchi isolated his bacterium granulosis came from Albuquerque, New Mexico, whither journeys were made by several American surgeons, and by Lindner to confirm the diagnosis of trachoma.

Therefore the trachoma studied by Noguchi does not differ from the trachoma found in Europe and in Egypt, at any rate clinically. The reviewer may note that Mayou, *Brit. Jl. of Ophthal.,* 1928, and Wilson, *Giza Lab. Reports,* Egypt, 1928, remark on the different histological features of trachoma in America and trachoma in Europe or Egypt. The principal difference is that the follicles in American-Indian trachoma consist almost entirely of epithelioid cells surrounded by a comparatively small ring of leucocytes. In European and Egyptian trachoma on the other hand the follicles are made up of lymphocytes and plasma cells, although there may be one or two epithelioid cells in old follicles. This is perhaps suggestive that there is more than one form of the disease.

The bacterium granulosis was passed by Noguchi through a series of monkeys, causing the development of follicles. The resulting condition was looked upon by Noguchi and the American surgeons as of a trachomatous nature.

Morax has carried out investigations on 11 cases of trachoma in exactly the same manner as described by Noguchi and with the same media but has been unable to detect an organism resembling the bacterium granulosis.

He then obtained from the Rockefeller Institute three ampoules containing Noguchi's bacillus. He found this to be a small Gram-positive organism, employing the staining technique of the Pasteur Institute. This was very surprising as Noguchi had described the bacillus as being Gram-negative. Correspondence with the Rockefeller Institute showed that the period allowed for decolouration was much longer there than at the Pasteur Institute.

As Morax considered that if the organism was Gram-positive it should be possible to find it in smears from trachomatous granulations he obtained from Algeria a large number of smears. In none of these cases was he able to find a Gram-positive bacillus resembling Noguchi's organism.

Morax found that the organism obtained from the Rockefeller Institute was easy to cultivate on ordinary media, and did not easily die out. He sent some of the cultures of bacterium granulosis to Tunis where Weiss of St. Louis inoculated four persons, though without producing the slightest pathological reaction.

The conclusion is that although Noguchi found a bacillus, not previously described, he did not find an organism causative of trachoma.

A. F. MACCALLAN.

(2) **Cuénod and Roger Nataf (Tunis).—Ultra virus et trachome.** *Rev. Internat. du Trachome*, January, 1930.

(2) **Cuénod and Roger Nataf** consider it advisable to suspend judgment as to the specificity of Noguchi's bacterium granulosis in the causation of trachoma. It is possible that this organism, though not itself causative, may perhaps be the carrier of an ultramicroscopic virus. We are reminded that Nicolle and Cuénod have shown that the pathogenic principle of trachoma is contained in a filtrable virus.

A. F. MACCALLAN

(3) **Ridley, Frederick.—Gas gangrene panophthalmitis.** *Trans. Ophthal. Soc. U.K.*, Vol. XLIX, 1929.

(3) **Ridley** gives a full account of a case of panophthalmitis due to the B.Welchii and of the bacteriological investigation by

which this diagnosis was established. The literature is reviewed—only five cases appear to have been reported—and these and the author's case are so similar in their main features that the following short summary of the condition as a clinical entity may be given.

Aetiology.—Perforation of the cornea or sclera by a foreign body which has been retained in the eye. In each case this has been a fragment of iron and the injury occurred when iron was being chiselled; in three cases contamination with earth was probable.

Bacteriology.—B. Welchii has been isolated in each case, streptococci and staphylococci have also been observed.

Morbid Anatomy.—The orbital oedema is not infective but is secondarily due to the panophthalmtiis. There is a wound of the cornea or sclera; chemosis is intense, the cornea is hazy and the anterior chamber may contain hypopyon; the iris is inflamed and may show a reddish discoloration. The tension, if the wound is closed, is high. On section (Chaillous) all the contents of the globe are disorganised and there is engorgement of the retinal and choroidal vessels, round which very few bacilli are to be seen, these being found in great numbers in the more central parts; the cellular reaction consists of polymorphonuclear leucocytosis.

Symptoms.—Early pain in the eye with loss of vision, followed by intense headache and collapse, which may be accompanied by vomiting. Temperature 100°—102°F. Onset of symptoms within twelve to twenty-four hours.

Locally.—Great swelling of the lids and adjacent parts which are dark red in colour and very tender; proptosis and fixation of the eyeball; chemosis and all the signs of an acute panophthalmitis. A bubble of gas, increasing in size under observation for a few hours, may be seen in the anterior chamber. Glycosuria may be present.

Diagnosis.—From orbital cellulitis—the history, the general condition of the patient, the temperature, the early onset of symptoms and the intense orbital oedema, with proptosis and fixation of the eyeball, are suggestive of the nature of the infection. If gas is present in the anterior chamber the diagnosis is strengthened; if the bubble of gas is seen to increase in size under observation it is confirmed.

Treatment.—The treatment adopted in this case consisted in excision of the centre of the cornea followed by evisceration, insertion of a drainage tube and irrigation with saline, avoiding injury to the sclera and conjunctiva. After-treatment included frequent irrigation with hydrogen peroxide and the administration

of anti-gas-gangrene serum. A drainage tube was retained until all discharge from the globe had ceased. The patient recovered and left hospital in ten days. It is uncertain whether extension of the infection into the orbit is to be feared if the orbital tissues are opened at operation but it is suggested that the treatment oulined is safe and effective.

Prognosis.—All cases have recovered.

A. F. MACCALLAN.

V.—THERAPEUTICS

(1) **Terrien, F. (Paris).—Ocular complications following the use of arseno-benzol compounds. (Des Complications oculaires après l'emploi des Arséno-benzols).** *Arch. d'Ophtal.*, September, 1929.

(1) The toxicity of the arsenical preparations employed in the early days of intravenous medication of syphilis has resulted for some years in their replacement by less noxious compounds. In spite of this change, and acquired experience in the administration of these drugs, ocular complications are not uncommon.

Terrien has observed a number of these unfavourable results in recent years, and this paper is based upon a clinical lecture given to his students to afford them some knowledge of the possible risks of this method of treatment.

Terrien considers these complications under two main headings: (1) Reactions which are purely toxic, affecting the normal eye and its adnexa, and shown chiefly by vaso-motor disturbance; they are not as a rule serious. (2) Early "specific reactions," by which he signifies reactions appearing soon after one or two injections; they are recognised to be of toxic origin, but are partially or wholly determined by specific lesions; they result perhaps from the sudden death of a large quantity of spirochaetes and the liberation of their toxins, or from an exacerbation of the virulence of spirochaetes preceding their destruction, or from crowding of them to the nerve centres when driven from the mucous and cutaneous tissues. The disappearance of the symptoms on continuation of the injections shows the specific nature of these reactions. Their site may be the cornea, uveal tract, retina, optic nerve, and exceptionally the motor nerves of the eyeball.

The paper is long and interesting and the subject well discussed. In his résumé Terrien urges great caution in the use of the arseno-benzols. He would reserve them for "certain forms of syphilis in

which mercury seems to exert an insufficient effect, a happening which is unusual when the drug is well employed, especially by intravenous injection."

The administration of the arseno-benzol preparation should be begun in small doses, 15 to 20 cgr., though even these amounts have given rise to grave crises. Finally if arseno-benzol medication is prescribed in cases of severe disease of the cornea or uveal tract which has proved resistant to other methods of treatment, it should never be employed if the optic tract is involved, particularly in the optic nerve affections of tabes; in such circumstances its use may be followed by rapid and severe aggravation of the disease.

J. B. LAWFORD.

(2) **Muncy, William M. (Providence, R.I.)—The treatment of gonorrheal ophthalmia.**—*Jl. of Ophthal. Otol. and Laryn.*, August, 1929.

(2) **Muncy** states that his only excuse for writing upon this topic is that during a practice of 21 years he has come to certain definite conclusions.

It is the predilection of the gonococcus to burrow between the cells into the deeper layers of the mucous membrane that accounts for their destruction and the difficulty of treatment.

Regarding actual methods of treatment the author commences by stating that nursing is not only the first, but the second and third most important factor.

Careful examination is the first step, and this should be carried out by the physician or house surgeon. The extent of invasion, and any corneal involvement is to be noted. Desmarres retractors should be used, and these should be lightly greased or dipped in water. For cleansing purposes, cotton dipped in boracic acid and squeezed between the fingers thus releasing a liberal stream of solution upon the eyeball, is used. On no account is rubbing, or a dropper to be used.

Actual treatment consists in irrigation of the conjunctival sac as fast as the material develops. This means in the active stage, every twenty minutes, increasing up to thirty minutes; later to be followed by hourly washings and when the discharge has abated, every two hours by day, and three by night.

If ice is used careful supervision should be exercised with regard to time employed. If the swelling of the lids is not controlled by ice in a few hours' application, then external canthotomy ought to be done.

Regarding the employment of antiseptics he has found argyrol the best and safest. Strengths under 25 per cent. are useless and the drug should be in constant contact with the infected area for

50 per cent. of the time. The preparation should be fresh and never used longer than fourteen days after preparation. Strengths of 25 per cent. to 40 per cent. can thus be used and are non-irritating. The eye is flooded with argyrol which is allowed to remain 20, 30, 60 minutes or even 1—2 hours depending upon the amount of discharge. After the next cleansing the eye is left free, until the next period when a fresh flooding is made. Thus whatever the frequency of cleansing the argyrol is present 50 per cent. of the time. If one eye only is affected the unaffected eye is treated with the same strength argyrol, three or four times in twenty-four hours.

If on the first examination grey exudate be noted on the cornea, however small, then milk injections or injection of similar proteid products (aolon) should be resorted to. Unless there is much swelling of the lids heat should be resorted to. Pledgets of cotton wool wrung out in hot water should be applied every two minutes at thirty-minute intervals. If an adult then the daily application of actinic rays from a quartz lamp is found to be most efficacious. This is applied by means of a quartz rod which is held $\frac{1}{8}$ of an inch from the ulcer for 20 seconds. Then treat the limbus nearest to the ulcer for 30 seconds. After the treatment one or two minims of 2 per cent. acriviolet are dropped upon the ulcer. This is slightly painful and can be preceded by holocaine or butyn. Cocaine must not be used. The acriviolet is applied once or twice daily.

A case is described of a woman aged 70 years, who contracted the disease from a grandchild. An annular ulcer of the cornea was present. Treatment consisted in argyrol 35 per cent., hot applications, and acriviolet. Milk injections were negative in this case. The result was excellent.

Attention is also drawn to the use of argyrol as a preventive measure in the newly born in place of the Credé method of 2 per cent. silver nitrate. The eyes are flooded with 35 per cent. argyrol which is allowed to stay for two hours. This to be repeated on second and third day. No infections have followed this treatment. The author does not state how long this technique has been in force.

He closes by impressing the fact that 75 per cent. of the care is nursing leaving argyrol in strong fresh solution flooded into the conjunctival sac 50 per cent. of the time, to do the rest.

S. SPENCE MEIGHAN.

(3) **Lemoine, A. N. (Kansas City, Mo.)—Ocular anaphylaxis.** *Arch. of Ophthal.*, June, 1929.

(3) **Lemoine's** paper opens with a brief survey of the work done on this subject, which was started by Uhlenhuth in 1903 when he showed that animals could be sensitised and immunised with lens

protein and that this hypersensitivity was tissue specific and not species specific. Elschnig showed the same thing with regard to uveal pigment in 1916 and suggested an allergic basis for sympathetic ophthalmitis, a suggestion which was elaborated by Alan Woods. Later Igersheimer laid down an anaphylactic basis for interstitial keratitis. Other lesions of the eye which have been attributed to anaphylaxis, comprise phlyctenular disease, recurrent intra-ocular haemorrhages and intra-ocular inflammations following rupture of the lens capsule (phako-anaphylactic endophthalmitis) also certain types of conjunctivitis, notably spring catarrh and a case of keratitis in a patient who proved to be hypersensitive to corn and was cured by desensitisation. The author then refers to a number of cases of his own. Thus : six patients who had conjunctivitis each summer were found to be sensitive to pollens, and the same was true of four cases of old trachoma who had recurrences each summer. One patient was hypersensitive to face powder and developed conjunctivitis from this cause, while of two cases of recurrent dendritic ulcer, one was found to be sensitive to chocolate and the other to pollens. Another interesting case was that of a man who had frequent attacks of diplopia with severe vertigo and teichopsia but no headache. The attacks ceased on removal of cheese from his diet and he never had another attack except after eating this food. There is another point, however, in the treatment of allergy, namely that the condition is often associated with endocrine imbalance, the thyroid, parathyroids and gonads being the glands most frequently involved. Thus the author has successfully treated some cases of spring catarrh by the administration of thyroid, parathyroid and calcium.

F. A. W.-N.

(4) **Beigelman, M. N. (Los Angeles).—The untoward effects of protein therapy in ophthalmic practice.** *California and Western Medicine*, p. 404, June, 1929.

(4) In this paper **Beigelman** discusses the dangers of protein therapy which treatment he has extensively used. Local complications are the results of imperfect technique but general and focal complications are more difficult to understand. Anaphylactic reactions are not common enough to warrant sensitization tests in each case and are usually well controlled especially with adrenalin.

The focal results of protein injection are not sufficiently understood yet but in cases where the part of repair is very low the injection may precipitate the most intense exacerbation in the inflammation under treatment. In spite of untoward results he in no way discredits the value of protein injections.

R. C. DAVENPORT.

VI.—GLAUCOMA

(1) **Calhoun, F. Phinizy (Atlanta, Georgia).—The vascular state and glaucoma.** *Amer. Jl. of Ophthal.*, April, 1929.

(1) **Calhoun** examined 64 patients with glaucoma in order to test the veracity of Charlin's observation that 90 per cent. of these cases show general vascular symptoms and that syphilis is almost always present in cases under 50 years of age. His conclusions were:—(1) Vascular disease was present in 95 per cent. of cases. (2) Syphilis was present in 15 per cent. of the vascular cases and in 14 per cent. of all cases, and nephritis in 40 per cent. and 37 per cent. respectively. (3) Abnormally high systolic pressure present in 42 per cent. and diastolic in 57 per cent. (4) Dilatation of the heart or aorta was present in 60 per cent. These observations lead the author to support Maitland Ramsay's suggestion that glaucoma and glomerular nephritis are both due to a toxin which produces dilatation of the capillaries and increased permeability of their walls.

F. A. W.-N.

(2) **Schoenberg, Mark J. (New York).—The artificial induction of ocular hypertension by compression of the jugular veins.** *Arch. of Ophthal.*, June, 1929.

(2) **Schoenberg** was induced to carry out the work detailed in this paper by the following experience. A patient had a tonometer applied to his eye with a 7·5 gramme weight. The pointer was deflected to the 6th division. Within a few seconds it had moved to the 5th and then to the 4th division. Schoenberg noted that the patient was sitting with his head bent so far back that his tight collar had produced a mild strangulation of his jugular veins, and his face was cyanotic. He therefore ascribed the rise of intra-ocular pressure to venous engorgement, and devised an apparatus for producing this condition in varying degrees. It consisted of a sphygmomanometer cuff, fitted with hooks and eyes so that it could be fastened round the neck and inflated, thus exerting pressure on the jugular veins. Three main types of reaction were obtained. In the first, the pressure rose during jugular compression and returned to normal after its release; in the second, it went down to lower than the initial figure after release of pressure; while in the third type, application of pressure to the jugular veins produced no alteration in intra-ocular tension.

F. A. W.-N.

(3) **Inman, W. S. (Portsmouth).—Emotion and acute glaucoma.** *Lancet, December* 7, 1929.

(3) It is difficult to give an abstract of **Inman's** short article, since, aside from the record of one particular and very suggestive

case, it contains a good deal of speculative matter which is not easy to synopsise. Briefly, Inman's suggestion is that while it is generally agreed that emotion as ordinarily understood may be the exciting cause of an attack of glaucoma, it may even be possible that " the onset may coincide with the anniversary of events once pregnant with emotion but now apparently indifferent or even long since forgotten." The author goes on to say, " The psychological investigation of such cases might lead to a further knowledge of the mechanisms involved, and so enable us sometimes to prevent the development of this very serious and painful disease by providing for the discharge of the emotion through legitimate paths." The reviewer confesses that he does not understand what is meant by " legitimate paths." He is not a psychologist and so may be quite mistaken in thinking that in order to lead a subconscious emotion along any path at all it would be only possible to do so at the risk of bringing the emotion acutely forward into consciousness. However that may be the case recorded is of interest, and should be read in the original. It cannot usefully be abbreviated. Further, the author is working at the subject more fully and it is hoped there may be other articles to follow.

ERNEST THOMSON.

(4) **Cantonnet, A. (Paris).—Ionisation in glaucoma.** (**l'Ionisation dans le glaucome.**) *La Clin. Ophtal.*, October, 1928.

(4) **Cantonnet's** article is not one which seeks to prove the fact or to discuss the details of ionisation in the case of the eye. It assumes as common knowledge that " medicamentous substances " penetrate the eye to such an extent that they can be found in traces in the urine. Ionisation of the eye reduces tension, temporarily it is true. The author has found it very useful in glaucoma, principally as a means of reducing the tension prior to operation in acute and subacute glaucoma. He employs chloride of calcium: the details are here given in French in order to avoid possibility of mistake in translation:—" chlorure de calcium 1/400, intensité 1 à 2/5 de milliampère " for 20 to 30 minutes, three or four times per day before opening the eyeball. In this way one can reduce the tension from 70 to 45 or 50 millimetres of mercury and thus operate under the best conditions. Then there are cases of acute glaucoma where operation is refused, and in such a case Cantonnet was able by means of pilocarpine and ionisation to reduce the tension and maintain " satisfactory " vision for three years. In chronic glaucoma, when operation is refused or when it is contra-indicated the procedure is as follows: " one gives ionisation *' séances '* of chloride of calcium if the tension is distinctly above normal, while iodide of sodium is employed if the tension is normal

or nearly so, in order to act upon the sclerosis rather than upon the tension." These ionisations are repeated every 4, 5 or 6 days so as to keep the eye in a favourable condition. The author is very careful to point out that he is not advocating ionisation as a substitute for operation. Where operation is decided upon and accepted; ionisation is a preparatory treatment. Only where operation is refused, or is contraindicated, does ionisation become a treatment *per se*.

ERNEST THOMSON.

(5) **Birch-Hirschfeld (Königsberg).—Tonometry in the diagnosis and the following up of glaucoma cases. (Zur tonometrischen Beurteilung des Glaukoms).** *Zeitschr. f. Augenheilk.*, Vol. LXX, p. 1, 1929.

(5) **Birch-Hirschfeld** relates his experience of three years' work on carefully recording the tonometric variations in over a hundred glaucoma cases. Readings were taken in these cases three times a day, at seven o'clock in the morning, at noon and in the evening. As is well known there is a considerable diurnal variation in the tension of the normal eye and according to Birch-Hirschfeld a disturbance in the normal rhythm of these variations is a symptom that precedes any other in glaucoma; the healthy eye gives variations akin to those of the temperature of the healthy body, whilst the fluctuations in the glaucomatous eye are more like the hectic fluctuations seen in a septic infection. In absolute glaucoma these hectic fluctuations may disappear, the tension remaining at a uniform high level, a parallel to the continuously high temperature sometimes seen in pneumonia. The use of miotics may favourably affect the fluctuations seen in glaucoma, but such effects are not necessarily permanent; it often happens that the hectic fluctuations typical of glaucoma return after a time. This should be considered a strong indication for operative interference. The return to normal after miotics or operation may be either by " crisis " or " lysis."

A point of considerable interest is the parallelism of the tension-charts of the two eyes. This is a process seen in normal eyes and persists in glaucoma, even when only one eye is affected. In such cases the sound eye will follow all the excursions, including any irregularities, of its glaucomatous partner, though of course, not to the same extent. This applies to primary glaucoma; in secondary glaucoma no such parallelism exists: the curves may even overlap, and this may also happen sometimes for a limited period after operation. Birch-Hirschfeld sees in this parallelism an argument in favour of a centre controlling ocular tension. The parallelism in the tension of the two eyes has a practical application, for the author has more than once avoided an operation on a definitely glaucomatous eye, by performing a decompression operation on

a practically blind fellow eye. The better eye shares the improvement in tension that the operation achieves in the worse eye; tonometric measurements clearly bring out the more regular variations in tension in both eyes as a result of an apparently needless operation.

"Tonometric measurements will become as indispensable in the treatment of glaucoma as the taking of the bodily temperature is in infectious diseases."

ARNOLD SORSBY.

BOOK NOTICES

Handbuch der speziellen pathologischen Anatomie und Histologie. Auge. Erster Teil. *Editor:* K. Wessely. *Contributors:* G. Abelsdorf, A. Elschnig, S. Ginsburg, R. Greeff, E. v. Hippel, W. Löhlein, F. Schieck. Berlin: Julius Springer. 628 illustrations. Pp. 1,042.

Two volumes of this System of Pathology are to be devoted to the pathological anatomy of the eye, and the book under review is the first of these. It comprises sections dealing with the conjunctiva, the cornea, the uveal tract, the retina, the optic nerve, the vitreous and glaucoma respectively, for each of which a different author is responsible. With trifling variations a similar arrangement of the subject matter is followed by each, a division of the material into subsections on a partly anatomical basis and a partly clinical one, having been adopted.

As the title implies, histological pathology is mainly dealt with, but some theoretical questions are briefly discussed—for example, the causes of retinal detachment and of glaucoma.

Bacteriology is included where its discussion is appropriate, and in some instances technical advice is given as to the best method of preparing the tissue under discussion for examination, while each of the first six sections opens with a description of the normal histology of that particular part of the eye. Slit-lamp observations are briefly referred to here and there, but the correlation of slit-lamp and microscopic findings is not a feature of the book.

At the end of each section a full bibliography is appended, and the general index leaves nothing to be desired in completeness. The illustrations are numerous and excellent; the great majority of these are reproduced from drawings, many of them coloured, and there are also a few microphotographs.

The actual text consists of a series of short résumés of the work of different authors of all nationalities (up to 1925 inclusive) the name of each being mentioned, and these résumés are interspersed

with introductory and descriptive remarks by the writers of the respective sections.

This treatise is the first work of its size and importance on the pathology of the eye which has appeared in any language for some years. It is in no sense a text book, but is essentially a work of reference, and in this capacity should prove especially useful, correlating as it does the results of histological research up to so comparatively recent a date.*

R. Clinica Oculistica di Roma, Diretta dal Prof. Q. di Marzio. Saggi di Oftalmologia. Vol. V. Anno 1929. Roma: Tipografia Poliglotta. Via Banchi Vecchi 12. Pp. 656.

This large and well printed volume corresponds to the English "Reports." The "samples" (*saggi*) cover subjects of all kind from radiotherapy to anisometropia. Many are of considerable interest and value. The paper by Neuschuler "On the behaviour of Descemet's membrane in trauma and inflammation" is a good attempt at systematising our knowledge of an obscure condition.

There is a short summary in English at the end of each article. Generally this gives the author's meaning well, but in one case (the paper of G. Caso on "Cylindroma of the supraciliary region") the summary states exactly the contrary of the author's intention. The tumour is a benign one "per mancanza di recidiva" etc.; in the summary "the case presented recurrence in a young subject": clearly a mistake for "occurrence."

Diseases of the Eye. MAY & WORTH. Sixth Edition. Pp. 475, with 22 plates. London: Baillière, Tindal & Cox. 1930. Price, 15s.

The authors have endeavoured to present a concise, practical and systematic manual of diseases of the eye intended for general practitioners and students. The book has been carefully revised but certain new methods of treatment, some of which may not survive the test of experience, are not discussed.

Mr. T. Harrison Butler has contributed a chapter on the slit-lamp and corneal microscope, and Mr. S. H. Browning has written an account of vaccine therapy in ophthalmology.

The first part of the book is devoted to a description of the methods of examination of the eye. The anatomy, physiology and pathology of each of the ocular structures is described seriatim. The chapters dealing with squint are extracts from "Squint: its causes, pathology, and treatment," by Claud Worth, sixth edition. These are particularly well written.

* On account of the importance of this book the opportunity was taken to review a purchased copy. The price paid for it was £13 8s., which we regard as an exorbitant price for a book of this kind.—EDITORS.

At the end of the book there is a chapter devoted to the ocular manifestations of general diseases, and another to ocular therapeutics, in which prescriptions of lotions, ointments, and other medicaments, are given in full.

The final chapter states the visual requirements for British and Indian Public Services.

There are many points of practical interest in this book which will meet the requirements of the general practitioner and the student.

CORRESPONDENCE

PROPTOSIS, PROTHESIS AND PROSTHESIS

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

SIRS,—It is very noticeable that a number of people, some teachers included, pronounce the word proptosis as if the two syllables were "prop" and "tosis." The word is, of course, "pro" and "ptosis," a falling forwards.

Then "prothesis" is a curious word. We use it commonly enough, but it is not advisable to employ it to a layman when we mean an artificial eye or a wooden leg, since its main dictionary meaning is "a place in a church on which the elements for the eucharist are put previous to their being placed on the altar." According to the Imperial Dictionary, "prothesis" has as its primary meaning the ecclesiastical one, and as its secondary meaning the surgical one, whereas "prosthesis" primarily has the surgical meaning and secondarily a philological one, *i.e.*, the adding of one or more letters to the commencement of a word such as *be*reft. It would therefore appear to be correct to refer to an artificial eye as a ***prosthesis*** rather than a prothesis.

Yours faithfully,

ERNEST THOMSON.

BLINDNESS IN INDIA

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

SIRS,—With reference to Col. Wright's communication on "Blindness in India" which appears in your April issue, may I request you to publish the following little explanation.

The Blind Relief Association was started by Mr. C. G. Henderson, I.C.S. in 1913, who, two years ago, gave up his appointment so as to be able to devote the whole of his time to this work. Last year, feeling the need for a more extensive effort the All India Blind Relief Association came into being to co-ordinate and extend the already existing Blind Relief Association.

This Association has never stated that it is the only agency for the prevention of blindness in India, and far from belittling the Government's effort, wishes whenever possible to work in conjunction with Government institutions. Its work is in rural areas, where no other agency exists for this one particular purpose, and where it is most needed. At present its activities are in the Bombay Presidency, which has a population of 26 millions and Government institutions dealing specifically with this problem number five only. Considering the fact that 80 per cent. of the population of India live in villages, which number more than half a million, the efforts of the Government can be said to have merely touched the fringe of the problem because its good work is mainly confined to towns and cities of the sub-continent which number 89 and constitute about 20 per cent. of the population. Urgent and absolute necessity has brought into existence the All India Blind Relief Association.

Yours truly,

B. P. BANAJI, F.R.C.S.I.
Hon. Ophthalmic Surgeon to the Association.

We have also received a letter on this subject, to a similar effect, from the Organising Secretary of the Blind Relief Association.—EDITORS.

NOTES

Honour

THE Leslie Dana Gold Medal, which is awarded annually in recognition of outstanding work on behalf of prevention of blindness, was presented on May 24, 1930, to Dr. G. E. de Schweinitz, of Philadelphia.

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Oxford Ophthalmological Congress

THE 20th Meeting will be held in Oxford on Thursday, Friday and Saturday, July 10, 11 and 12.

There will be a discussion on Toxic Amblyopia and Retrobulbar Neuritis, introduced by Dr. Traquair, Sir Farquhar Buzzard, Bt., K.C.V.O., and Professor J. A. Gunn, on Thursday morning.

In the afternoon Sir Oliver Lodge, F.R.S., will give an Address entitled "Modern Theories on the Nature of Light." On Friday morning the Doyne Memorial Lecture will be delivered by Dr. Harry Friedenwald, of Baltimore, on "Pathological Changes in the Retinal Blood Vessels in Arterio-sclerosis and Hypertension." In addition, Professor Schüller, of Vienna, will give an address on "Recent Developments in Radiology associated with Ophthalmology." In the afternoon an address will be given by Professor Gonin, of Lausanne, on "Retinal Detachment and its Treatment." Papers will also be given by Dr. Samuels, of New York, Mr. Harrison Butler, Dr. de Courcy and Dr. Van Heuven, of Utrecht.

Ophthalmologists desirous of attending the Congress are requested to communicate with the Hon. Secretary, C. G. Russ Wood, 12, St. John's Hill, Shrewsbury, who will send application forms for Membership and furnish all particulars.

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The Chinese Cornea

THE *Lancet*, referring to an article by Dr. Pillat in the *National Medical Journal of China*, for October, 1929, says, "Dr. Pillat calls attention to a peculiarity in Chinese eyes compared with European, in that the upper limbus is frequently exaggerated in sickle form above the cornea so that the shape of the anterior corneal surface is elliptical with a white crescent above, which by the novice may be taken for an arcus senilis, and which affects the landmarks for incision into the anterior chamber when operating for cataract or glaucoma."

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Section of Ophthalmology, Royal Society of Medicine

ELECTION of Officers and Council for 1930-1931: The following have been nominated :—*President:* E. W. Brewerton. *Immediate Past President*: (ex-officio) : Cyril H. Walker. *Vice-Presidents :* A. H. Levy, A. E. J. Lister, W. H. McMullen, O.B.E., Ransom Pickard, C.B., A. Hugh Thompson, A. J. Ballantyne. *Hon. Secretaries*: P. G. Doyne, O. Gayer Morgan. *Other Members of Council :* (Physicians) : Wilfred Harris, W. J. Adie. (Metropolitan Members) : N. Bishop Harman, J. Cole Marshall, Rupert S. Scott, J. F. Cunningham, O.B.E., W. S. Duke-Elder, J. D. M. Cardell, C. L. Gimblett, Humphrey Neame, M. H. Whiting, O.B.E. (Provincial Members) : J. Burdon-Cooper, H. M. Traquair, T. M. Bride, W. Clark Souter, D. M. McKay, J. W. Tudor Thomas. *Representative on Library Committee (Honorary Librarian of the Bowman Library):* F. A. Williamson-Noble. *Representative on Editorial Committee:* Robert C. Davenport.

FUTURE ARRANGEMENTS

1930

July 10-12.—Oxford Ophthalmological Congress, at Oxford.
August 26-29.—British Medical Association, at Winnipeg.
October 7.—Midland Ophthalmological Society, at Birmingham.
December 2.—Midland Ophthalmological Society, at Birmingham.

CONTEMPORARY OPHTHALMIC LITERATURE

American Journal of Ophthalmology. *May, 1930.*

MADDOX. A new 'eye and hand' method of training squint.
WILDER. Trachoma among the Indians.
BEDELL. Traumatic rupture of the choroid with detachment of the retina: spontaneous re-attachment.
LLOYD. Tonometry and the various instruments employed in its practice.
MCKEE J. W. Melanosarcoma at the limbus.
VELEZ. Optic nerve surgery.
GLOVER. Some eye observations in tuberculous patients.
ROWLAND and ROWE. Concentric contraction of the visual fields.

Annales d'Oculistique. *April, 1930.*

GABRIÉLIDÈS. Cytodiagnosis of epithelioma or glioma of the retina.
ZAMENHOF. The intrinsic shadows in the fundus.
SUBILEAU. Haemoaspiration and trephining with the aid of the electric burr in dacryocysto-rhinostomy.
SÉDAN. Development of four spontaneous fistulae after iridectomy for acute glaucoma
SALVATI. A new procedure for the correction of cicatricial entropion.
BERNEA and SEGALLER-MIRON. Practical conclusions on aseptic and antiseptic technique in eye operations.

Archives d'Ophtalmologie. *May, 1930.*

MAJEWSKI. The mechanism of detachment in myopes.
ROLLET. Extirpation of an orbital hydatid by subaponeurotic orbitotomy.
WEEKERS. Treatment of painful eyes retaining vision by orbital injection of weak alcohol.
PATRY. Medical examination of motoris's.

Revue d'Oto-Neuro-Ophtalmologie. *February, 1930.*

REBOUL-LACHAUX and BRAVINSKY. Algo-myoclonic encephalitis with high hyperalbuminosis and ocular myoclonus.
BEAUVIEUX. Transitory hemianopias from angio-spasm.
ABADIE and LAUBIE. Divergent strabismus from permanent dissociated paralysis of the 3rd nerve in a post encephalitic Parkinsonism.
REBIERRE. Static crises in an old case of contusion and wound of the eye.

March, 1930.

ANDRÉ-THOMAS, SCHAFFER and VEIL. Associated paralysis of conjugate movement in Parkinson's disease.
PESME, BOISSERIE-LACROIX and PHILIP. A case of post-rubeola bilateral ophthalmoplegia of nuclear origin.

Archiv für Ophthalmologie. *April, 1930.*

BRUNNER. On the mode of transmission of the various forms of congenital red-green blindness,
SCHEERER. On the topography of the edge of the optic nerve in myopic and non-myopic eyes.
KRAVKOV. On central influence on visual acuity.
ZAMENHOF. A new method of investigation of the fundus in focal illumination.
THIES. Iritis and menstruation.
KESTENBAUM. On the development of the eye movements and of optokinetic nystagmus.
OHM. On our knowledge of nystagmus.
WÄTZOLD. Further researches on the pathogenesis and clinical picture of choroidal sarcoma.
TOWBIN and RAWIC-SCERBO. On the question of the relationship of scrofulous kerato-conjunctivitis to tuberculosis.
BRAUN. The valuation of Mantoux's intradermal tuberculin reaction as a guide to the diagnosis of tuberculous eye affections.
KREIKER. On the clinical picture and pathology of epithelial xerosis of the conjunctiva.
v. HIPPEL. On the prognosis of uveal sarcoma.

Zeitschrift für Augenheilkunde. *May, 1930.*

SAFAR. Injection of alcohol in the treatment of blepharospasm and spastic entropion.
SAUER. Combination of disturbance of accommodation and convergence after encephalitis.
LEWITSKY. Technique of the determination of the near point.
CAVKA. Electric cataract with cyclitis.
FALTA. On the physiological connection between the centre for convergence and the centre for lateral movements of the eyes.
BULAC. A case of perforating wound of the cornea from a chip of stone.

Bolletino d'Oculistica. *April, 1930.*

ALAJMO and ACCARDI. Alcoholic-tabetic toxic amblyopia.
SALA. The action of radiant heat in the treatment of trachomatous conjunctivitis.
GUALDI. Voluntary lacrimation with hypertrophy of the palpebral lacrimal glands.
BERGER. Renal function and cataract.
BELLAVIA. On the influence of extract of ciliary body on the size of the pupil, the ocular tension and the index of refraction.

Archivos de Oftalmología Hispano-Americanos. *May, 1930.*

VERDAGUER. Spring catarrh and Hebra's prurigo.
MENDOZA. Double astigmatism in the clinic.
ARRUGA. New model of blepharostat to avoid pressure on the globe during intra-ocular operations.

Revista Oto-Neuro-Oftalmologica y de Cirugia Neurologica. *April, 1930.*

PIRES. Optic neuritis and acute ascending paralysis of Landry.

Revista Cubana de Oftalmologia. *March, 1930.*

FERRER. Dacryocystitis neonatorum.
CASTRESANA. Surgical treatment of concomitant convergent strabismus.
VALASCO. The pathogenic theories of glaucoma.
STINCER. Cerebral tumours.
AVALOS. Disturbances of the cervical sympathetic leading to ocular changes.